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In Memoriam

RUDOLPH MATAS (1860-1957)

On September 23, 1957, the world lost one of the most eminent pioneer thoracic and vascular surgeons by the death of Rudolph Matas. Dr. Matas was a founder member of The American Association for Thoracic Surgery and its president in 1921. Although Dr. Matas will be remembered largely for his work in vascular surgery, particularly in the field of aneurysms, he will also be remembered for his original investigative work in thoracic surgery, being one of the first to emphasize the disastrous effects of acute surgical pneumothorax. At the meeting of the Southern Surgical and Gynecological Association (now the Southern Surgical Association) in 1899, Dr. Matas reported on intralaryngeal insufflation (Intralaryngeal Insufflation for the Relief of Acute Surgical Pneumothorax: Its History and Methods With a Description of the Latest Devices for This Purpose, J.A.M.A. 34: 1371, June 2, 1900) and stated: "Until the danger of seriously interfering with respiratory functions by introducing acute collapse of the lung is thoroughly eliminated or is reduced to a safe minimum, an analogy between the pleura and the peritoneum from the surgical point of view will never exist." In May, 1898, he discussed surgery of the chest and emphasized the value of the Fell-O'Dwyer apparatus as a method of preventing and overcoming the effects of acute traumatic thorax in thoracic operations and, in a presentation before the American Surgical Association in May, 1901 (Artificial Respiration by Direct Intralaryngeal Intubation With Modified O'Dwyer Tube and a New Graduated Air Pump: Its Application to Medical and Surgical Practice, Tr. Am. S. A. 19: 392, 1901), he reported the results of animal experimental investigations, which he and Dr. John Smyth performed, demonstrating that a pump employing negative as well as positive pressure was actually harmful. He stated: "After demonstrating to our satisfaction that the double inspiratory and expiratory pump was not a practical appliance for artificial

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respiration, we abandoned the aspirating cylinder entirely in our work and utilized a single insufflating cylinder on the O'Dwyer principle and found it perfectly reliable and made it the basis for our practical clinical respiratory machine."

Dr. Matas was born in Bonnet Carre, Louisiana, a short distance from New Orleans, on September 12, 1860. His father and mother, who were natives of the Spanish province of Catalonia, returned to Spain shortly after young Rudolph's birth. In 1867, they left Barcelona and returned to the United States, where Dr. Matas had his preliminary education in New Orleans and in Matamoros, Mexico. He enrolled in the Medical School of the University of Louisiana (now Tulane University) in the fall of 1877 and received his Doctor of Medicine degree in March, 1880. While still an undergraduate medical student, he won competitively a two-year internship at the Charity Hospital and also, while a medical student, was appointed to the Havana Yellow Fever Commission as an interpreter and laboratory assistant. He went with the Commission to Cuba in 1879. In 1880, while an intern at the Charity Hospital, he assisted at the first laparotomy performed in that institution under Listerian ritual, including the use of carbolic acid spray. In 1882, shortly after graduation, he went to Mier, Mexico, to treat an epidemic of yellow fever in that city. In 1886, he was appointed Demonstrator of Anatomy at Tulane University, holding this position until he was appointed Professor of Surgery and Chairman of the Department of Surgery in 1894. On his retirement in 1927, he became Professor of Surgery Emeritus.

Dr. Matas's first endoaneurysmorrhaphy was performed on May 16, 1888, when, after attempting to control an aneurysm by proximal and distal ligation, he found there was bleeding through many collaterals which he was able to demonstrate by opening the aneurysm. Following removal of the tourniquet, he saw that blood was coming from various orifices and reasoned that suturing the orifices might control the bleeding, which it did; thus, the operation of endoaneurysmorrhaphy was accomplished. In commenting on this operation when he saw the well patient 10 years later, he stated: "At the time this operation was performed, I had no precedence to guide me. The only work that had been done was that of Glueck, published in Langenbeck's Archives, in which he detailed nineteen experiments in the suture of arteries which had failed to a great extent in spite of the ingenious methods that he devised. The exigencies of the case, however, made it necessary that hemostasis should be obtained, and the occlusion by suture appeared to be so easy and plain that it seemed to me that any surgeon similarly situated would instinctively have adopted this simple way of getting out of the difficulty." His experience in the treatment of aneurysms was enormous because of the great wealth of material at the Charity Hospital, both in spontaneously occurring and traumatic aneurysms. He also did considerable experimental investigative work, and he and Dr. Carroll Allen devised an aluminum band which permitted temporary occlusion of vessels without injury to them. Before the American Surgical Association in St. Louis on May 2, 1940, Dr. Matas reported a total of 620 operations for aneurysms, of which 101 had been treated by the suture method. Another contribution

which Dr. Matas made to vascular surgery was elaboration of the hyperemic test to determine the adequacy of collateral circulation following obliteration of the vessel (Test to Determine the Efficiency of the Collateral Circulation Before Attemping Permanent Occlusion of the Great Surgical Artery, Ann. Surg. 52: 126, 1910).

He did more than anyone else to elucidate our concept of the production of elephantiasis and its prevention. His first discussion on this subject was before the Southern Surgical Association in 1911, and his extensive report in 1913 (The Surgical Treatment of Elephantiasis and Elephantoid States Dependent Upon Chronic Obstruction of Lymphatic and Venous Channels, Am. J. Trop. Dis. and Prevent. Med. 1: 60, 1913) is a classic today. Dr. Matas also performed the first Kondoleon operation in the Western Hemisphere. The original communication by Kondoleon appeared in the *Zentralblatt für Chirurgie* on July 27, 1912, and Dr. Matas's first Kondoleon operation was performed on October 20, 1912.

Dr. Matas had tremendous interest in the increasing incidence of thromboembolism. In a report before the International Society of Surgery in Warsaw in July, 1929, and in the Donald Balfour Lecture at the University of Toronto on April 5, 1932, he emphasized that this was a disease of modern surgery.

Dr. Matas is known for several "firsts." He was the first person to use intravenous saline in a surgical patient. He was also one of the first to advocate gastric drainage and, in 1924, emphasized the value of the indwelling Jutte tube, introduced through the nose. Dr. Matas was one of the first to use spinal anesthesia and on November 18, 1899, he first used cocaine. He did considerable investigative work in the anatomic laboratory to determine the method and best site of injection in spinal anesthesia. He devised an apparatus for infiltrative anesthesia. He was the first person to differentiate between typhoid fever and malaria in Louisiana.

Dr. Matas was one of the twelve founders of the American College of Surgeons in 1913, was president of the College in 1925-1926, and was awarded an honorary Fellowship by the College in 1952. He was president of the American Surgical Association in 1909, president of the International Society of Surgery in 1936, president of the Southern Surgical and Gynecological Association (now the Southern Surgical Association) in 1910. In 1938, he was awarded the first Certificate of Merit by the American Medical Association. He was a member of practically every surgical association in the world and was an honorary member of most of them. Among his honorary memberships were those in the Royal Academy of Medicine of Barcelona, the Royal College of Surgeons of England, the National Order of Carlos Finlay, the Order of Leopold of Belgium, the Gorgas Medical Society, the Havana Academy of Medicine, and the Spanish Royal Academy. He was made a Knight of the Legion of Honor of France (Chevalier Legion d'Honneur) in 1932, and was given the rank of Officer in 1950. He received the Henry Jacob Bigelow Medal in 1926, and, in 1940, received the *Times-Picayune* Loving Cup as the most eminent citizen of New Orleans for that year.

Dr. Matas was a great student and had an unprecedented fund of information. He was said by the late Dr. Will Mayo to be the world's best educated physician. One could not help but be impressed by Dr. Matas's sincerity, extreme courtesy, modesty, and gentleness. If, however, courage and firmness were needed, he was amply endowed with both. In speaking of the medical student who faints when he sees his first operation, he stated: "The youth who feels and struggles at his first surgical experience shows that he is normal, that he is psychically and physiologically sound, he shows that he has feeling, that he has a human quality that will make him a humane surgeon. He is a man who will worry and who will, through his very sensibility, feel most keenly the burden of his responsibility when it will be his turn to be a leader and a master. It is all wrong for people to believe that the sight of blood is indifferent to the surgeon. On the contrary, the older and more experienced he is, the more anxious he is to conserve it and to see as little of it as possible in the course of his operation. Every real surgeon is happy when he can speak of his bloodless operation, and he is miserable when he has to admit that he has spilled much of it in an unavoidably bloody one."

Although Dr. Matas retired shortly after the founding of The American Association of Thoracic Surgery, he always maintained a keen interest in the Society and its activities as well as in the tremendous developments made in thoracic and cardiovascular investigations. We as an organization have greatly benefited because of his contributions and activities.

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Original Communications

INTRATHORACIC PHEOCHROMOCYTOMA

Including a Case of Multiple Paragangliomas of the Functional and Nonfunctional Type

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AWARENESS that a pheochromocytoma may occasionally occur within the thorax is of importance from two aspects. If the surgeon fails to recognize the nature of such a tumor when found in the chest, serious or even fatal complications may occur due to improper management of the patient from whom such a neoplasm is being removed. On the other hand, the investigation of a patient with paroxysmal or sustained hypertension must include roentgen studies of the chest since an intrathoracic pheochromocytoma or related type of tumor may be responsible for the blood pressure elevation. In some patients more than one pheochromocytoma may be present and one of these tumors may be located within the thorax.

Since those pheochromocytomas which occur in the usual sites in and around the adrenals cannot be visualized radiographically without special techniques, most retroperitoneal pheochromocytomas are only discovered by special roentgenograms or surgical exploration after the patient has developed intermittent or sustained hypertension and special tests have indicated that the hypertension is due to abnormally large amounts of circulating norepinephrine and epinephrine. By contrast, however, we have removed an intrathoracic pheochromocytoma from 3 patients in whom the paravertebral mass was first seen roentgenologically at a time when hypertension was not manifest. One of these patients was known to have had the tumor 3 years before hypertension was demonstrated. A second patient had the asymptomatic intrathoracic pheochromocytoma removed following the previous excision of a hypertension-producing perirenal

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pheochromocytoma. The third patient had no definite hypertension but the mass was demonstrated on a routine chest roentgenogram and the biologic activity of the excised pheochromocytoma was proved by chemical analysis.

The recent literature attests to the increasing awareness of the importance of pheochromocytomas. Yet in many reports insufficient emphasis is accorded to the relatively uncommon locations in which this tumor may occur. Often discussion is limited to various diagnostic procedures which may aid in the localization of the lesion in the adrenal, perirenal, and retroperitoneal areas. More emphasis should be given to adequate roentgen examination of the thorax since a pheochromocytoma in the chest is not as rare as the literature to date would suggest. The fact that several cases have been seen in one large city and yet only a few others reported from other parts of the world lends support to this belief.

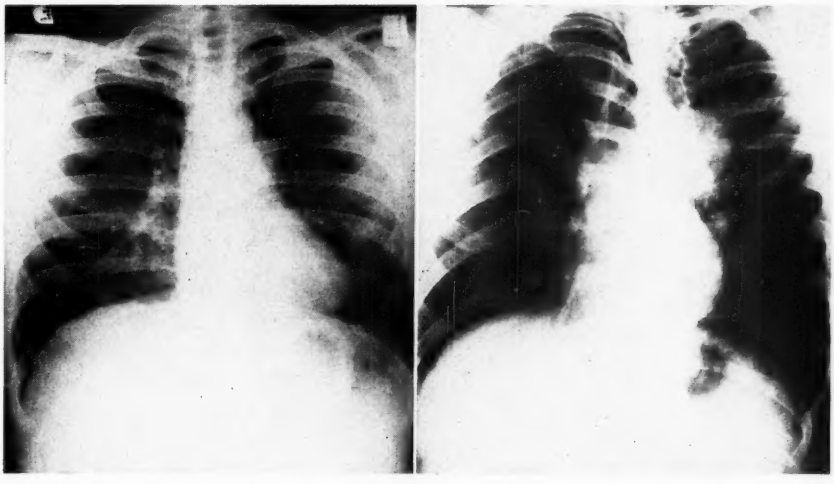
As pheochromocytomas occur in the course of the chromaffin system, the intrathoracic tumors have usually been found along the sympathetic trunk in the paravertebral region just lateral to the heads of the ribs. Such a tumor is located retropleurally. Usually the growth is a small mass which may be flattened out against the inner aspect of the chest wall in the costovertebral groove.

Roentgen demonstration of an intrathoracic pheochromocytoma may not be readily made unless the tumor is large. Since the size of a pheochromocytoma may not necessarily be closely correlated with the degree of its biologic activity, a small tumor may nevertheless be very important from the clinical standpoint. Radiographic examinations should be done with the following points in mind. On a posteroanterior film the growth may be obscured by those mediastinal structures which project laterally beyond the lateral margins of the vertebral column. Even an aortic knob or the shadows cast by the pulmonary hilum may hide the pheochromocytoma by superimposition of shadows. Obviously the cardiac silhouette may completely obscure a paravertebral tumor of even considerable size if reliance is placed entirely on a single posteroanterior roentgenogram. The lateral film may not give the help which it often does in costovertebral tumors because the pheochromocytoma is often a rather flat tumor which may not cause recognizable density in the costovertebral area. In one of our cases the lesion could not be seen on the lateral film even in retrospect. Films taken in both oblique projections may be most helpful in demonstration of a small costovertebral tumor, provided overexposure of the film is avoided.

There are several gross anatomic features of an intrathoracic pheochromocytoma which should alert the surgeon to the possibility that the tumor which he has exposed may be a paravertebral pheochromocytoma. The first thing noted may be the marked vascularity of the pleura overlying the lesion. Numerous small vessels in the tissue adjacent to the tumor may even suggest the possibility of a hemangiomatous growth. The profuse vascular supply of the small mass may be further demonstrated by the amount of bleeding which follows incision of the pleura even at a slight distance from the tumor. The intrathoracic pheochromocytoma is usually found to lie in the course of the sympathetic chain as do certain other neurogenic neoplasms. The gross appearance

of the pheochromocytoma suggests a soft glandular growth rather than the usual more firm consistency of most neurogenic tumors. The pheochromocytoma is often a dark reddish brown in color.

When the surgeon encounters a mass with such gross characteristics of a pheochromocytoma, he should be particularly alert if the patient has ever had blood pressure readings of a fluctuating type or shown hypertension. But even in the absence of such evidence the possibility of pheochromocytoma may not be dismissed as one of our cases demonstrates. Changes in the circulatory status of a patient with a pheochromocytoma may occur so rapidly during manipulation and excision of the tumor that preparedness is essential. The surgeon



A. *B.*

Fig. 1.—Case 1. *A*, Posteroanterior roentgenogram of chest shows a small rounded mass projecting from the mediastinal shadows just below the arch of the aorta.

B, Oblique view shows mass in left costovertebral region extending from the fifth to the seventh ribs.

should inform the anesthetist of the possibilities and request a very close observation of the circulatory status, with blood pressure readings every few minutes during the remainder of the operation. Norepinephrine should be made immediately available in case hypotension develops after occlusion of the blood supply of the tumor. Manipulation of the growth should be kept to a minimum so as to lessen the liberation of pressor substances into the blood stream with a possible hypertensive crisis. Phentolamine (Regitine) should be given in adequate doses if much elevation of blood pressure occurs. Pathologic examination of the excised tumor by immediate frozen section is advisable. By this means a pheochromocytoma may be distinguished from other neoplasms found in the thorax. The surgeon is then better able to guide the patient's postoperative therapy.

CASE REPORTS

CASE 1.—(Previously reported by H. C. M.[8]) This man had a costovertebral tumor demonstrated on a chest roentgenogram when he was 22 years old and asymptomatic. Three years later he complained of fatigue, nervousness, palpitation, occasional dizziness, headaches, and sweating. The blood pressure was then between 210/120 and 190/100 mm. Hg. The roentgenograms showed no change in the size of the tumor (Fig. 1). At operation the possibilitiy of a pheochromocytoma was considered and frequent blood pressure readings taken.

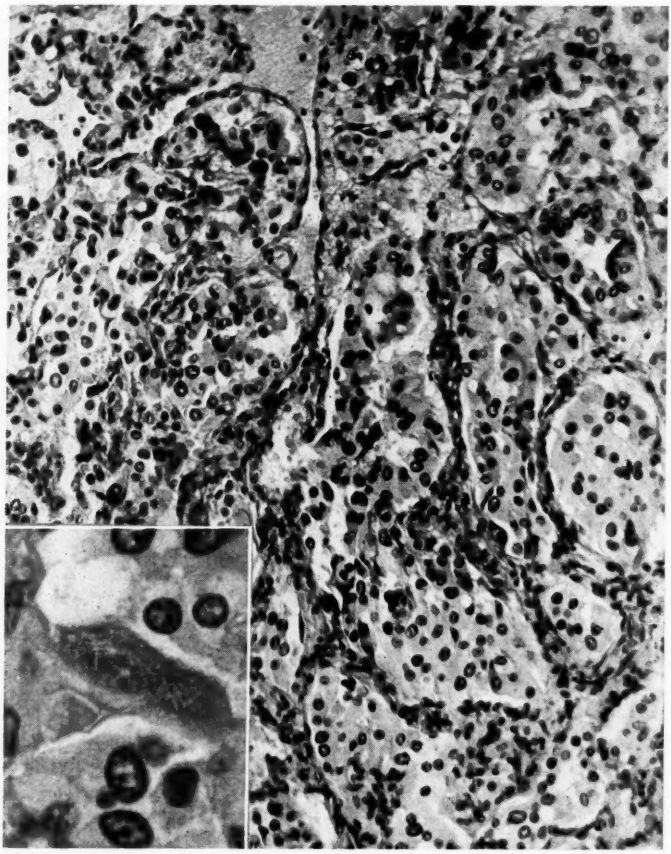


Fig. 2.—Case 1. Photomicrograph of pheochromocytoma. The characteristic arrangement of tumor cells in globules separated by capillaries is shown. Insert shows chromaphilia granules.

The blood pressure dropped precipitously just after the tumor was removed but responded somewhat to Adrenalin. Hypotension persisted during the first postoperative day. The blood pressure then rose again to a range between 180/110 to 160/100 mm. Hg. Not until 2½ years later was the blood pressure normal. The patient was well and normotensive 10 years later. Pathologic examination of the tumor revealed a pheochromocytoma (Fig. 2).

Comment: This case suggests that the patient with a pheochromocytoma may change from an asymptomatic status to a symptomatic hypertensive state without obvious

enlargement of the tumor. Excision of a pheochromocytoma may sometimes be followed by a gradual return of the blood pressure to a normal level although usually a rather rapid decrease occurs.

CASE 2.—J. C. (L.H.H.184471), a 48-year-old man, had no complaints when he was examined in a cancer detection clinic in November, 1954. Physical examination was essentially negative. Blood pressure was 120/76 mm. Hg. Roentgenogram of chest showed a rounded mass with smooth, well-defined, outer border located at the level of the right hilum, measuring about 5 cm. in diameter (Fig. 3). The lesion could not be definitely located on the lateral view. It was at first incorrectly assumed that the density was situated near the ascending aorta. On fluoroscopy, the posterior location of the mass was detected later.

When seen by one of us (H. C. M.) in January, 1955, a history was obtained of an occasional pain radiating down from the right lower thoracic region to the abdomen. A neurogenic tumor was suspected and operation recommended.

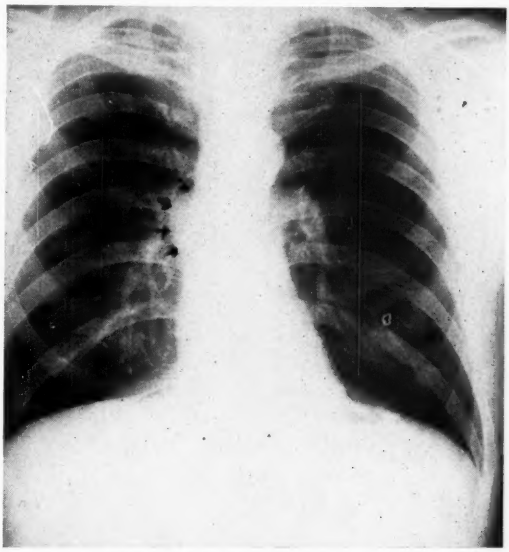


Fig. 3.—Case 2. Posteroanterior roentgenogram shows a small rounded mass (indicated by arrows) projecting out slightly from the mediastinum at the level of the right hilum.

At thoracotomy a soft, slightly lobulated tumor was found in the right costovertebral area at the level of the seventh and eighth thoracic vertebrae. The tumor was retropleural and flattened out against the chest wall; the sympathetic trunk traversed the mass. A number of blood vessels entered various portions of the tumor. The possibility of a pheochromocytoma was considered when the tumor was exposed because of the similarity to the appearance of the tumor in Case 1. Frequent blood pressure readings were taken as the growth was excised but no significant deviations occurred. Considerable bleeding was associated with removal of this 4 by 5 by 1.5 cm. pheochromocytoma.

The postoperative course was uneventful and there were no significant blood pressure changes.

Pathologic examination revealed a pheochromocytoma. No mitoses were seen. Occasionally tumor cells were seen in the lumen of blood vessels. Schmorl stains gave a positive chromaffin reaction.

The tumor specimen was analyzed by Dr. Marcel Goldenberg and found to contain 2.8 mg. of norepinephrine per gram of tumor tissue, and nonmeasurable traces of epinephrine.

The patient was well 39 months after operation.

Comment: This case might have been considered as a nonfunctioning paraganglioma if chemical analysis of the tumor had not been made. The gross appearance at operation was the first time that the diagnosis of pheochromocytoma was considered; this suspicion was confirmed by immediate pathologic examination. This case illustrates the fact that a functioning pheochromocytoma can be present in the absence of hypertension. It is not justified to conclude that the absence of hypertension rules out pressor activity.

CASE 3.—P. D. (P.H.844975.) This patient at the age of 8 years had first noted some difficulties in vision, but ophthalmological examination was normal. During the next several years there were episodes of emotional instability and variable visual disturbances. On Oct. 4, 1946, at the age of 11, he was admitted to the Columbia-Presbyterian Medical Center (Babies Hospital) following ophthalmological examination which revealed retinal exudate, and a blood pressure of 260/190 mm. Hg. The eye findings in this case have been reported by Bruce.[2]

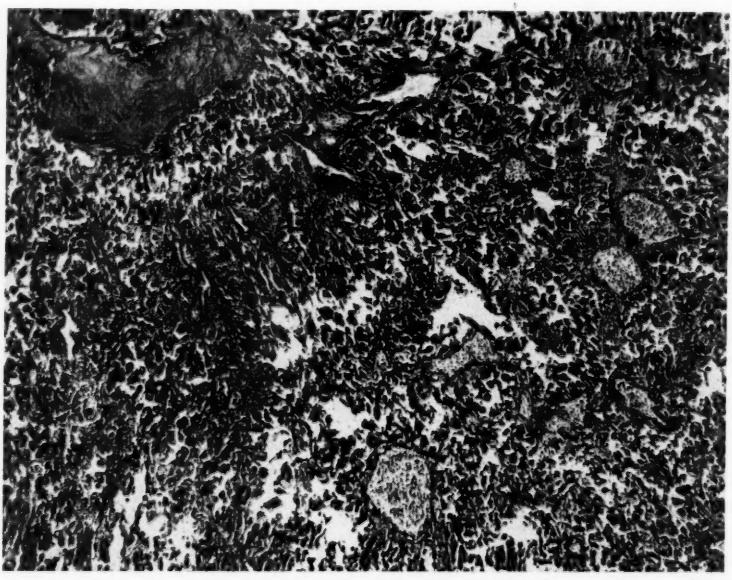


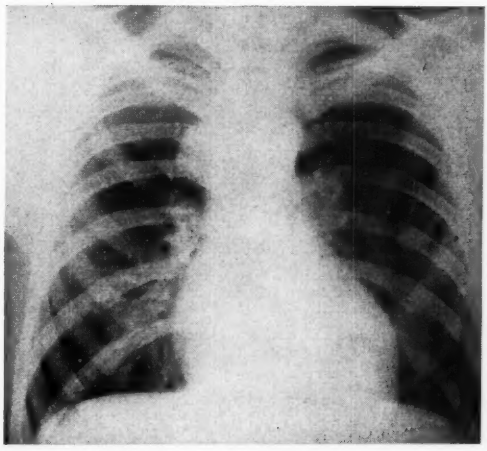
Fig. 4.—Case 3. (G.U. 7409—X-133.) Photomicrograph of the partly degenerated retroperitoneal tumor removed from the renal area in October, 1946. The organoid paraganglioma tous pattern is easy to recognize.

During work-up he had episodes of severe pounding headache, pallor, sweating, abdominal pain, and nausea, during which blood pressure rose to 270/210 mm. Hg, with a pulse rate of 150. Benzodioxane tests showed a drop of blood pressure to 138/110 lasting only a few minutes, but subsequent to this there was a slow and progressive fall in blood pressure. Pyelograms showed evidence of hypoplastic right kidney.

First operation: Oct. 29, 1946 (Dr. George Cahill). Operation revealed a globular tumor, 4.5 by 4 by 2 cm. in dimensions, attached to the right renal vessels and closely applied to the inferior vena cava. The right kidney was hypoplastic, the right adrenal normal. The kidney and tumor were removed together. During operation, blood pressure rose to 160/110 mm. Hg during manipulation of the tumor, fell to 85/50 just after the tumor had been removed, and stabilized at 110/80 at the close of the procedure. Pathologic study revealed a pheochromocytoma with extensive central necrosis (Fig. 4).

Following operation, his pressure remained normal at approximately 120/80, his eye signs regressed, and he recovered without complication, being discharged on Nov. 12, 1946, 14 days after operation.

Two years later, in Sept., 1948, at the age of 13 a routine x-ray of the chest revealed a rounded shadow of homogeneous density and smooth border projecting from the mediastinum in the right posterior chest at the level of the sixth rib (Fig. 5). He was admitted to the Columbia-Presbyterian Medical Center (Presbyterian Hospital) on Dec. 6, 1948. At this



A.

B.

Fig. 5.—Case 3. *A*, Posteroanterior roentgenogram showing paraspinal thoracic pheochromocytoma at T_5 and T_6.
B, Lateral roentgenogram showing paraspinal pheochromocytoma.

time he appeared to be entirely well and asymptomatic. The nephrectomy wound was well healed, his blood pressure was 110/70 mm. Hg, blood count and urinalysis were not remarkable.

Second operation: Dec. 8, 1948 (Dr. George Humphreys). A soft, extremely vascular tumor about 5 cm. in diameter was found protruding from the posterior chest wall between the fifth and sixth ribs. It apparently rose from the fifth dorsal ganglion since the sympathetic chain entered it at its upper pole and left at its lower pole. It was closely applied to the azygos vein and numerous large thin-walled veins ran from the azygos and the adjacent intercostal vessels over the tumor. All vessels surrounding the tumor were first ligated, after which the tumor was separated from the fifth and sixth ribs to which it was closely applied.

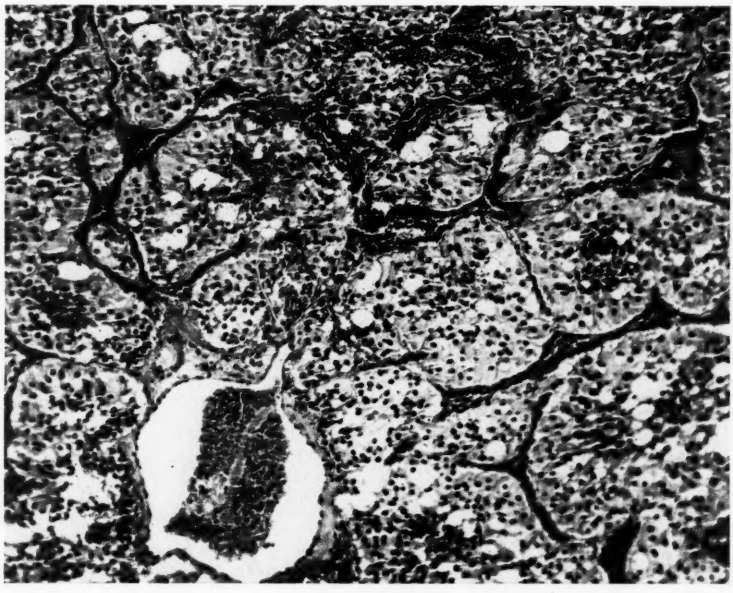


Fig. 6.—Case 3. (A-10001—X-133.) Photomicrograph of the paragangliomatous tumor removed December, 1948, from the paravertebral area at the level of the fifth dorsal sympathetic ganglion.

There was no evidence of extension through the intervertebral foramen. During manipulation of the tumor, blood pressure rose from a preoperative level of 90/60 to 160/80 mm. Hg, falling again at the close of the procedure to 90/50. Pathologic examination revealed a 27-gram pheochromocytoma of typical histology (Fig. 6).

He made an uneventful recovery and was discharged on Dec. 17, 1948, 9 days after operation. For 5 years he remained well. In January, 1954, he developed a mass in the right lateral neck.

Third operation: Feb. 7, 1954, at Hackensack Hospital. A well-encapsulated tumor was removed which proved on section to be a nonchromaffin paraganglioma of carotid body type (Fig. 7).

Three years later, on July 24, 1957, he was again admitted to the Columbia-Presbyterian Medical Center (Neurological Institute) complaining of progressive numbness of the legs, loss of control in walking for 2 months, and back pain for 3 days. X-rays showed evidence of a destructive lesion of the fifth thoracic vertebra and physical examination showed evidence of cord compression at this level (Fig. 8).

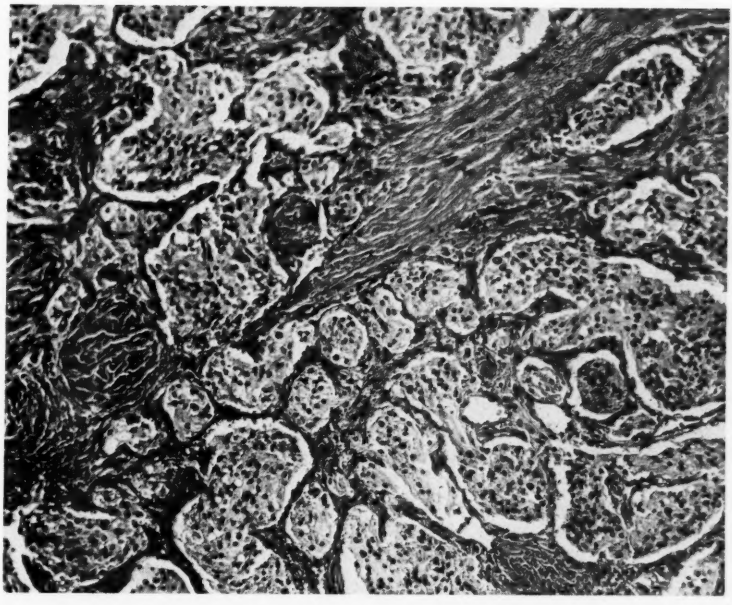


Fig. 7.—Case 3. (P&S 44313—X-133.) Photomicrograph of the paraganglioma removed from the bifurcation of the right carotid artery in March, 1954.

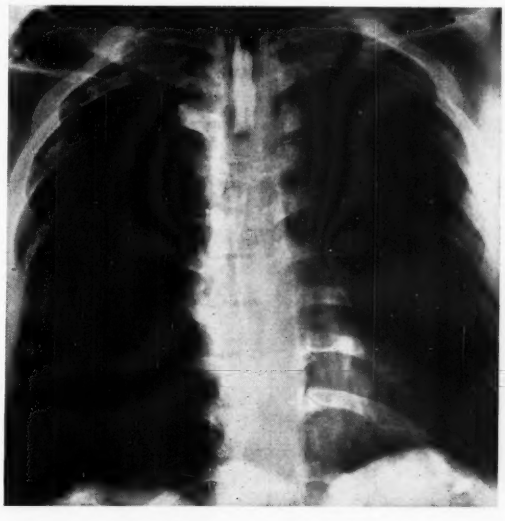


Fig. 8.—Case 3. Myelogram, August, 1957, showing upper limit of intraspinal tumor and regeneration of fifth rib removed 9 years previously. Bony destruction of the pedicle of the fifth dorsal vertebra is present but not well seen in this view.

Fourth operation: Aug. 5, 1957 (Dr. James W. Correll). There was severe spinal cord compression throughout the area of the laminectomy which extended from the second to the seventh thoracic vertebra. The tumor was found to overlay the spinal cord dorsally, to extend to the right and around the spinal cord ventrally. The tumor was entirely in the epidural space. The dura was not opened. At the termination of the procedure, the spinal cord was well decompressed with the tumor extruding through the area of the laminectomy and allowing the spinal cord to return toward its normal position. The tumor was extremely vascular. Excision of a small segment resulted in a copious loss of blood. Since the tumor extended ventral to the spinal cord and since it involved the vertebrae and mediastinum, no attempt at wide excision was made. Hemostasis was achieved with electrocautery and Gelfoam. Blood pressure remained steady throughout the procedure at 140/80 mm. Hg, except for a short period when (presumably due to blood loss) it fell to 110/80, returning with transfusion to the original level at the close of the procedure. The histologic diagnosis was paraganglioma of the extradural space.

Following operation, temperature rose to 103° F. for 2 days, then fell to normal, and he recovered without further complications.

X-ray therapy was begun at the Neurological Institute by Dr. Juan Taveras and completed at the Hackensack Hospital by Dr. Eric J. Ryan. At the Neurological Institute, 1,200 r in air were given over the dorsal spine from Aug. 15 to Aug. 21, 1957. At the Hackensack Hospital 1,500 r in air were given from Aug. 22 to Aug. 29, 1957.

Following operation, the patient showed evidences of progressive improvement. With the assistance of physiotherapy, he rapidly became fully ambulatory. Convalescence was complicated by homologous serum jaundice treated at the Hackensack Hospital. Fortunately, this was mild. In November, when last seen, he was carrying out all his usual activities and was preparing to return to the religious organization to which he belongs. At this time, neurological examination showed no evidence of abnormality. A satisfactory course continues.

Comment: This case is extraordinary in several respects. The patient has developed three (and possibly four) independent tumors of the paraganglionic type. The first was a functional chromaffin paraganglioma or pheochromocytoma associated with the right kidney. The second was an intrathoracic tumor of the same type originating in the fifth dorsal sympathetic ganglion. The third was a nonchromaffin paraganglioma of the right carotid body. Nine years after removal of the intrathoracic pheochromocytoma, an intraspinal tumor developed at the same level. This was also a paraganglioma, presumably chromaffin. Although extension into the foramen was looked for and not seen at the time of removal of the thoracic tumor, it is quite possible that the intraspinal tumor was a "dumbbell" extension of the thoracic tumor. It is unlikely that it arose from a dorsal root ganglion as an independent tumor. Multiple primary paragangliomas have been described[4] but the finding of both nonchromaffin and chromaffin types in the same patient is very rare.[12] Since the last tumor was not removed, it is probable that the final chapter in this case is not yet complete.

REVIEW OF LITERATURE

The first intrathoracic pheochromocytoma, an incidental finding at autopsy in a 39-year-old woman, was reported by Miller[11] in 1924. The tumor was in the right costovertebral area at the level of the sixth rib, and was about the size of two walnuts. The report did not state whether the patient had had hypertension.

The second report of an intrathoracic pheochromocytoma also diagnosed at autopsy was by Philips[15] in 1940. This man was 39 years old at the time of death, but was known to have had hypertension since the age of 29 years. He complained of episodes of cardiac palpitation and throbbing in his head, as

well as some dyspnea, polyuria, and nocturia. At 33 years of age he experienced a left hemiplegia. The heart was enlarged and the blood pressure varied between 170 mm. Hg and 220 mm. Hg systolic; while the diastolic pressure level was about 120 mm. Hg. Roentgenographic examination of the chest showed a mass at the left apex posteriorly, which did not move on swallowing and did not compress the trachea. The patient also had a Horner's syndrome on the left side. Six years later the patient had an acute coronary occlusion and died in congestive heart failure. At autopsy, a firm mass about the size of a plum was found at the apex of the left pleural cavity. The mass was not attached to the lung and was easily shelled out. In addition to the tumor, the autopsy revealed myocardial infarction and marked arteriosclerosis of the coronary arteries, aorta, and renal vessels.

Overholt, Ramsay and Meissner[14] reported a case of an intrathoracic pheochromocytoma in a 16-year-old boy who had no known hypertension prior to operation. Chest roentgenograms had demonstrated a paravertebral tumor on the left side of the chest in the region of the fourth to the seventh vertebral bodies. Other than mild heat intolerance the patient was asymptomatic and his preoperative blood pressure was 110/75 mm. Hg. During the early portion of the operation which was undertaken with a preoperative diagnosis of neurofibroma, the blood pressure rose to 240/160 mm. Hg and the presence of a pheochromocytoma was suspected. The systolic blood pressure remained at 180 mm. Hg until the blood supply to the vascular tumor was interrupted; then there was a drop in blood pressure to 90/70 mm. Hg but additional blood and Neosynephrine elevated the pressure to a systolic level of 170 mm. Hg. Due to the vascularity of the tumor, a total of 1,500 c.c. of blood was given during the operation. Nevertheless, in the early postoperative period the blood pressure again declined to a systolic level of 40 mm. Hg and, although there was again a response to Neosynephrine, the pressure was in the range of 70 mm. Hg systolic during most of the first 24 hours. The patient then made an uneventful recovery and had a normal blood pressure upon discharge from the hospital. Pathologic examination revealed the typical findings of a pheochromocytoma but no biologic assay of the tumor was performed.

Nissen[13] reported on a case of hypertension in a 22-year-old woman. A thoracolumbar sympathectomy was undertaken and, during the operation on the first side, a pheochromocytoma was found along the thoracic sympathetic chain and removed. The blood pressure became normal following this operation and was still normal 2½ years later.

Cone and associates[3] recently reviewed 34 reported cases of pheochromocytomas in children. In one of their cases a chest roentgenogram had shown a paravertebral mass in the right side of the chest in the region of the seventh and eighth thoracic vertebrae which was proved at postmortem examination to be an intrathoracic pheochromocytoma. The child died following operation for massive intestinal bleeding. Autopsy also revealed a pheochromocytoma in each adrenal gland. The intestinal hemorrhage had apparently been due to an occlusive endarteritis with infarction of the intestine secondary to the marked

atheromatous changes produced by the severe hypertension which this cachectic 8-year-old patient had manifested. The pheochromocytoma in the thorax weighed 20 grams and contained 1.8 mg. of norepinephrine per gram of tumor tissue. This was a somewhat lower norepinephrine content than was found in each adrenal tumor. This patient's mother and younger sister also had a pheochromocytoma. In children, the incidence of bilateral and extra-adrenal pheochromocytoma is more common than in adults. Of 34 cases reported in children, a tumor was in the right adrenal in 13, and in the left adrenal in 8, in both adrenals in 9, and extra-adrenal in 7. Thus, a total of 46 tumors was found in the 34 children. The intrathoracic pheochromocytoma reported by Cone[3] is the first reported case of an intrathoracic pheochromocytoma in a child.

McLeish and Adler[10] reported a case of intrathoracic pheochromocytoma with hypertension in a 19-year-old male who noted pain in the left side of the chest following exercise. The pain was usually preceded by nausea. The patient also complained of sweats, tachycardia, faintness, and headaches. Roentgenograms showed a small tumor at the seventh rib on the left side. Cure followed successful removal of a 26-gram tumor.

Apparently the secretion of pressor amines is not limited to pheochromocytomas because Mason and co-workers[9] demonstrated noradrenaline in a thoroughly documented case of intrathoracic neuroblastoma. Their patient was a 5-month-old baby girl who had attacks of pallor, sweating, and rapid breathing lasting a few hours with apparent well-being at other times. Roentgenograms demonstrated a large rounded mass in the costovertebral area. During the attacks the blood pressure was elevated and fever was sometimes demonstrated; the attack could be abruptly ended by intravenous phentolamine. There was an increased amount of pressor amines in the urine, especially during one of the attacks. A preoperative diagnosis of intrathoracic pheochromocytoma was made and thoracotomy revealed a pink tumor arising from the left sympathetic chain. During and immediately after operation the characteristic blood pressure fluctuations, such as are encountered in the removal of an actively secreting pheochromocytoma, were observed. Microscopic examination of the tumor, however, showed a characteristic neuroblastoma and a lymph node contained a metastasis. Postoperatively the blood pressure fell to a normal range in 1 week and no further attacks of pallor, sweating, or dyspnea occurred. The child was well 1 year after operation. Assay of the 50-gram tumor showed it to contain 0.14 mg. of pressor amines per gram of tumor.

Wahl and Robinson[18] reported a case of neuroblastoma of the mediastinum with pheochromoblastomatous elements. The patient, a 4-year-old boy, died of a neuroblastoma in the upper posterior thoracic region which had metastasized diffusely. Microscopic examination of the primary tumor revealed a sympathogonioma with mixed neuroblastic and pheochromoblastic elements. Transition could be seen between primitive nerve cells and the chromaffin cells. The metastases were all of the embryonic neuroblastomatous type whereas the differentiated elements were present only in the primary growth. The child's blood pressure was not mentioned.

Von Euler and Strom[17] reported a series of cases of pheochromocytoma and included 1 case of a 31-year-old man with multiple tumors in the thorax and persistent hypertension.

In addition to the cases cited above, there are almost certainly others which have been reported as paragangliomas. Godwin and associates[5] reported a case of paraganglioma of the upper posterior mediastinum. The patient had a normal blood pressure but the gross and microscopic description of the tumor seem similar to the findings in a pheochromocytoma.

DISCUSSION

Roentgen demonstration of a tumor along the course of the thoracic sympathetic chain in a patient with hypertension requires consideration of the diagnosis of pheochromocytoma. Determination of the urinary catechol excretion is then in order. In some cases of intrathoracic pheochromocytoma, the growth may be detected radiographically before hypertension or other clinical evidence of its chemical activity may be apparent. In such cases the urinary catechol excretion may be normal. Our cases demonstrate that a change in size of the tumor may not necessarily accompany the transition from a non-hypertensive to a hypertensive state. But a normal blood pressure does not necessarily mean that the pheochromocytoma does not contain pressor substances. Chemical analysis of such tumors will further elucidate the correlation between the clinical and chemical findings.

It is beyond the scope of this paper to give a lengthy discussion of the large variety of symptoms which may occur in patients with a pheochromocytoma. Although, in most cases, hypertension of some type is a significant finding, the absence of hypertension in some patients with a chemically active pheochromocytoma has already been indicated. A patient with a pheochromocytoma which secretes sufficient norepinephrine and epinephrine to produce symptoms may manifest certain signs and symptoms not usually found in patients with essential hypertension. Excessive sweating, peripheral vasomotor phenomena due to vasoconstriction, elevated body temperature, slightly elevated fasting blood sugar, somewhat elevated basal metabolic rate, tachycardia, and glycosuria may be present. It can readily be seen why such patients may be confused with cases of hyperthyroidism and mild diabetes mellitus.

Pheochromocytomas are frequently multiple, therefore the demonstration of a pheochromocytoma in one of the common locations does not preclude the presence of another tumor within the thorax. The tumor may show familial occurrence. It is sometimes associated with neurofibromatosis. Malignant metastases may also be actively secreting.

In recent years, additional laboratory methods of detecting the presence of a functioning pheochromocytoma have been developed. These tests depend on the quantitative determination of the pressor amines in the blood or urine by fluorimetric analysis. Elevated levels of epinephrine and norepinephrine in the blood are usually demonstrated if the patient has a sustained hypertension but normal concentrations of pressor amines may be found in some patients

with paroxysmal hypertension unless the test is done at the time of a spontaneous or induced elevation of blood pressure. Elevated concentrations of pressor amines in the blood may also be found in some patients with renal insufficiency, increased intracranial pressure and lymphoma in the absence of pheochromocytoma. The elevation of the catechol level in the blood of some cases of uremia is due to impairment in the renal secretion of these substances. It might be expected, therefore, that severe renal disease associated with a pheochromocytoma might cause a reduction in the catechol output in the urine.

In some cases, the augmented secretion of catechol amines occurs intermittently but in the majority of cases it is continuous. According to von Euler and Strom,[17] there is a good agreement between the proportion of norepinephrine and epinephrine in the tumor and in the urine. While norepinephrine is increased in the urine in most all cases of secreting tumors, epinephrine is increased only in certain cases. In no instance in their series was an increased content of epinephrine alone found in the urine or tumor. Von Euler and Strom have investigated the possibility of localizing a pheochromocytoma by the catechol amine level in samples of the venous blood taken by catheter in various portions of the caval system in a case of multiple, possibly malignant, intrathoracic pheochromocytoma; samples from the inferior caval vein and the branchiocephalic veins did not contain norepinephrine but a high concentration appeared in the superior vena cava at the level where the azygos vein enters.

The body position of the patient during operation and the choice of anesthetics may be of importance for the catechol amine release from the tumor. The urinary output of catechol amine should be determined after operation in order to ascertain the completeness of the surgical treatment. A high arterial blood pressure after an apparently successful operation may be due to an essential hypertension existing before the presence of the tumor, or to vascular changes secondary to the period of elevated blood pressure caused by the tumor, or may be produced by remaining undetected tumor tissue elsewhere.

Von Euler and Strom[17] reported on the norepinephrine and epinephrine content of 15 pheochromocytomas removed from patients with hypertension. The norepinephrine content varied from 0.2 to 8.4 mg. per gram of tumor. In 7 cases, the norepinephrine was less than the 2.8 mg. per gram found by Goldenberg in our case without hypertension but, in most of these, the tumor was larger and thus the total norepinephrine content was greater. Thus, a relatively high norepinephrine concentration may be present in a small tumor without hypertension being manifest at the time. The epinephrine content of a pheochromocytoma is usually considerably less than the norepinephrine concentration. According to von Euler and Strom, the pheochromocytoma producing only norepinephrine is usually situated more or less away from the adrenal glands. Our intrathoracic pheochromocytoma which was analyzed by Goldenberg showed only nonmeasurable traces of epinephrine.

The anesthetic management of the patient with known or suspected pheochromocytoma requires special consideration.[1] In the selection of anesthetic

agents, drugs which render the patient particularly vulnerable to circulatory depression would seem to be inadvisable but, on the other hand, an excitement stage would appear to be highly undesirable because it might well cause an undue outflow of pressor substances from the pheochromocytoma and thus result in an hypertensive crisis. Cyclopropane would seem to be inadvisable because of the possibility of predisposing to cardiac irritability in the presence of a high pressor level in the blood. Spinal anesthesia would not be considered for an intrathoracic pheochromocytoma and would seem undesirable anyway. The chief objectives in anesthetic management seem to be a smooth induction unassociated with any hypoxia or hypercarbia.[16]

The anesthetist must take very frequent blood pressure readings because of the rapid fluctuations which may occur. Regitine should be given if the blood pressure becomes unduly elevated during the period before the pheochromocytoma is removed. After excision of the tumor, when a precipitous drop in blood pressure frequently occurs, large amounts of norepinephrine may be required to maintain the blood pressure at a satisfactory level. These patients may require amounts of norepinephrine which would be highly excessive for individuals not previously adapted to high blood pressor levels.

The literature attests to the great danger of a surgical procedure performed for some other disease upon a patient with an unsuspected pheochromocytoma. The magnitude of the operative procedure may not be of great importance because severe and fatal hypertensive and hypotensive crises have occurred with anesthesia and relatively minor surgical procedures. In any patient who manifests such unexplained circulatory derangement the possibility of an undiagnosed pheochromocytoma must be entertained. The roentgenogram of the chest must be properly scrutinized for any evidence of even a small costovertebral mass as a part of the total investigation although it is far more likely that a pheochromocytoma, if present, would be located in or near the adrenals.

Some authors have employed the term paraganglioma for pheochromocytomas which occur outside the adrenals. It seems illogical, however, to utilize a different term for a tumor of the same histologic and biologic characteristics merely because it occurs in a different anatomic location. Moreover, these tumors are derived from the chromaffin system whether they are in the adrenal area or arise in other parts of the body. One reason for the designation of the extra-adrenal pheochromocytomas as paragangliomas is the recognized fact that the presence or absence of a positive chromate reaction may depend in part on the fixing and staining technique employed. However, the significant factor is whether or not the tumor produces norepinephrine or epinephrine or both. It is this biologic characteristic which sets these tumors apart. But since these pressor substances may be present in the tumor without hypertension being manifested by the patient, it is obvious the chemical analysis of the neoplasm is the final criterion in doubtful cases. Thus, some tumors reported as paragangliomas in normotensive individuals may actually have been pheochromocytomas.

An analysis of the literature on pheochromocytoma gives multiple indications that these tumors have been frequently missed on routine postmortem examination. The difficulty in finding the tumor when it is of small size is well illustrated by 1 case in which seven surgical procedures were undertaken before the pheochromocytoma was finally located.

In 1951, Graham[6] reported on an analysis of 207 cases of pheochromocytoma with hypertension collected from the literature. In addition he encountered 41 reports of cases of tumor in which there was little or no clinical data and 32 cases of the neoplasm with no evidence of hypertension. Since that report, many additional cases have been published. It is evident that an increasing awareness of the possibility of pheochromocytoma accounts for the more frequent reports in recent years. Moreover, an increasing number are being recognized in childhood and a greater number in unusual locations are being reported. In the past few years several cases of pheochromocytoma in the urinary bladder, which resulted in hypertensive episodes associated with micturition, have been diagnosed.

The nonchromaffin paragangliomas found in the chest include tumors arising from the aortic bodies.[7] These rare lesions are not associated with hypertension. In reference to our Case 3, with carotid body tumor and pheochromocytoma, it is interesting to note that some workers have reported the extraction of a blood pressure lowering substance from a carotid body tumor. Thus, the question has arisen whether, when both types of tumors are present in the same patient, each might produce an antagonistic effect on the secretion of the other.[12]

SUMMARY

Paroxysmal or sustained hypertension in a patient with a tumor in the costovertebral region of the thorax should lead to the consideration of an intrathoracic pheochromocytoma. When operative manipulation of a soft vascular costovertebral tumor produces considerable fluctuation in blood pressure, even though no preoperative hypertension was recorded, the surgeon must consider the possibility of a pheochromocytoma and take precautions to avoid hypertensive and hypotensive crises by appropriate drug therapy during operation. Since pheochromocytomas may be multiple, an intrathoracic location must be considered as a possibility. Adequate roentgen examination of the chest combined with urinary catechol excretion determinations are the principle diagnostic aids. Surgical excision can effect a cure if instituted sufficiently early and with proper safeguards based on recognition of the nature and behavior of the tumor.

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(For Discussion, see page 653)

THE MANAGEMENT OF STAPHYLOCOCCIC TENSION PNEUMATOCELES BY INTRACAVITARY SUCTION TUBE DRAINAGE

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WHEN infant lung tissue is infected with staphylococcic organisms, the pattern of inflammatory response predisposes to various tension phenomena within the pneumonic area. The multiple small abscess cavities represent defects in the pulmonary parenchyma subject to the disruptive elasticity of surrounding intact lung. The small bronchioles are filled with plastic exudate and cellular debris so that they function as valves, permitting air to enter the diseased tissue with each inspiration but limiting expiratory deflation. Tension areas cannot decompress by the mechanism of collateral ventilation because the surrounding lung is consolidated.

Frequently this sequence results in pleural perforations causing tension pyopneumothorax. It is also the cause of the large pneumatoceles commonly seen during convalescence. Although pneumatoceles would appear to be sequelae of the pneumonic episode (being most impressive in roentgenograms exposed during convalescence), they do appear on occasion during the acute pneumonic episode. The high incidence of early tension pyopneumothorax would suggest that the train of pathologic events which ultimately causes pneumatoceles is initiated with the primary necrotic infection and that pneumatoceles might be expected in any phase of the illness. They must either be obscured or actually prevented from expanding by pneumonic consolidation and by pneumothorax compression of the potential cystic spaces.

Although the pneumatoceles may assume alarming dimensions they have little influence upon the patient's well-being in the convalescent period—usually disappearing in time without treatment. However, the effect of a tension pneumatocele appearing in the phase of acute pneumonia may be quite different. Infants in the acute stages of staphylococcic pneumonia are desperately ill, and the slighest derangement of a precarious ventilatory reserve may be lethal.

A consideration of the mechanism of production of pneumatoceles in this illness suggests that the actual amount of lung tissue damaged is slight and that the large size of the cystic area is the result of ballooning of small necrotic foci acted upon by positive pressure gas forces. For this reason and since

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pneumatoceles are known to disappear over the course of months, leaving a normal-appearing lung, this would appear to be the situation par excellence for the utilization of conservative intubation methods in an effort to preserve a maximum degree of lung function. It must be clearly demonstrated that the course of the pneumonia is being influenced adversely by early ballooning of such a pneumatocele and, consequently, that some form of surgical treatment is necessary to preserve life.

As we observed an increasing number of infants with staphylococcic pneumonia on our Dependents' Service at the Chelsea Naval Hospital after 1956, we encountered a rare situation in which two pneumatoceles caused ventilatory

Fig. 1.—Inflammatory changes in staphylococcic pneumonia predisposing to the later development of tension cysts. The intense inflammatory response has resulted in the formation of two microscopic abscess cavities (*A*). The small bronchus communicating with the lower abscess is undergoing necrosis and is filled with cellular debris and exudate (*B*).

distress in an infant during an acute pneumonic episode. Suction drainage was used to tide this infant over the critical period. It proved to be the only treatment necessary for management of the air cysts. At that time we were not aware of any other application of intracavitary suction drainage in this disease. Recently, numerous excellent articles on staphylococcic empyema have brought up the possibility of applying suction tube drainage to staphylococcic pneumatoceles. A precise documentation of its actual application and value, however, has not appeared. It is likely that Wallman, Godfrey, and Watson[1] intubated cysts in 2 infants in the acute stages of staphylococcic pneumonia, although their description does not permit us to draw conclusions as to the exact nature of the procedure. Recently, Rakower and Wayl[2] reported that staged Monaldi drainage caused the resolution of a pneumatocele treated some time after subsidence of pneumonia.

Fig. 2.—Resolution of pneumatoceles without treatment.
Left: Bilateral pneumatoceles appeared as the pneumonic process subsided.
Middle: Although the pneumatoceles in the right upper lung field distended enough to cause mediastinal displacement 25 days later, the child was experiencing only slight dyspnea on exertion and consequently was managed conservatively.
Right: Two months later, the cysts were resolving. Patient was asymptomatic. Cysts completely disappeared in subsequent roentgenograms.

CLINICAL EXPERIENCE LEADING TO THE APPLICATION OF INTRACAVITARY SUCTION

Between January, 1956, and April, 1958, we treated 12 infants with pneumonia attributable to staphylococcic infection. In each instance, the organism could be cultured from blood, empyema fluid, sputum, or throat swabbings. The last-mentioned culture source was reliable in indicating the offending pathogen since each of the 12 patients presented characteristic signs of staphylococcic disease: empyema, tension pyopneumothorax, pneumatocele, or a combination of such complications. Bacteriologic studies demonstrated the expected resistance to penicillin, streptomycin, and the tetracyclines, with sensitivity to chloramphenicol, erythromycin, and Albamycin. Analysis of hemolytic activity and coagulase activity was incomplete. Phage typing was not carried out.

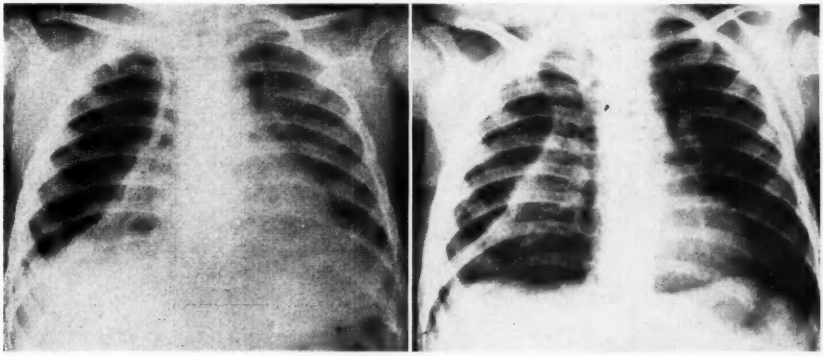

Fig. 3.—Differentiation of an atypical pleural pocket from pneumatocele.
Left: A tension pneumothorax may be misinterpreted as a pneumatocele by an inexperienced observer. A small pneumatocele in the collapsed right lung demonstrates the characteristic rounded appearance and surrounding lung tissue, features not evidenced by the elongated tension pneumothorax.
Right: A later film demonstrated expansion of the right lung posterior to the pyopneumothorax pocket. The pneumatocele is now irregular in appearance, probably as a result of resolution of bronchial inflammation and decompression of the cyst.

The 12 patients ranged in age from 1 week to 6 years. Eight were under 1 year of age. No sex predilection was apparent in the small series; 7 of the patients were males and 5 were females. Pyopneumothorax was the sole pleural complication, occurring in 7 of the 12 patients. All of the patients with empyema had an associated air leak. Two patients with small accumulations of air and exudate could be managed by needle aspiration alone. This was utilized as the sole means of treatment only when toxicity was slight, the pockets were small, and accumulations were slow in developing. The sustained maintenance of negative intrapleural pressure after aspiration was an indication for the continuing application of needle aspiration. If any suggestion of toxicity or rapid accumulation of fluid or air was observed, trocar intubation was carried out, connecting the No. 16 Fr. catheter to suction of −10 to −15 cm. of water. One patient did not come under our care until a small pocket of fluid and air was well on the way to resorption without the aid of needle aspiration. Since pneumatoceles subsequently developed, we have listed the pleural complication in this case as pyopneumothorax, although we lack aspiration evidence

TABLE I

CASE	AGE AND SEX	CULTURE SOURCE	PLEURAL COMPLICATIONS		PNEUMATOCELES				
			TYPE	TREATMENT	NUMBER	PNEUMONIA	TREATMENT	DAYS, ONSET OF PNEUMONIA TO APPEARANCE	MONTHS, ONSET OF PNEUMONIA TO DISAPPEARANCE
1	3 mo. F	Throat	Pyopneumothorax	None	2	RML	Observation	13	2½
2	19 mo. M	Empyema	Pyopneumothorax	Intubation	0	L RUL	---	--	--
3	2 mo. M	Blood	None		2	LLL	Observation	29	6½
4	9 mo. F	Throat	Pyopneumothorax	Needle aspirations (multiple)	1	RLL	Observation	24	1½+
5	2 mo. M	Empyema	Pyopneumothorax	Intubation	0	RLL	---	--	--
6	17 mo. M	Empyema	Pyopneumothorax	Needle aspirations (3)	3	RUL RLL	Observation	20	1
7	3 yr. F	Throat	None		1	RLL	Observation	11	1+
8	6 yr. M	Throat	None		2	LLL RUL	Observation	19	2+
9	2 mo. F	Throat	None		2	RLL LUL	Observation	10	2+
10	1 wk. M	Empyema	Pyopneumothorax	Intubation	0	R L	Dead		
11	2 mo. M	Throat	None		3	RUL RML RLL	Observation	17	11 days+
12	2 mo. F	Empyema	Pyopneumothorax	Intubation	4	R	Intubation	14	2

as to the character of the fluid. Intrapleural tubes were removed when the lungs were expanded, pressure fluctuations were slight, and only after 24-hour periods of clamping were not associated with reappearance of air or fluid.

It was interesting to note that the 9 infants in whom pneumatoceles developed showed roentgen evidences of cyst formation early, within 10 to 29 days after onset of the pneumonia. In most of the 9 infants, pleural complications and toxicity from the pneumonic process were well on the way to resolution once the pneumatoceles appeared. However, in 1 case, the period of toxicity was prolonged and the infant showed pleural disease and ventilatory disturbance as a result of ballooning cysts during the acute illness.

This patient was treated successfully by intracavitary suction tube drainage. We did not consider tube drainage necessary in any of the other patients in whom pneumatoceles developed. In several of these patients, the cysts assumed alarming proportions on the roentgenogram and, in 1 infant, dyspnea was attributable to cyst distention. However, the pneumatoceles resolved in every instance as we patiently adhered to the doctrine of masterful inactivity.

Of the 4 patients in whom pyopneumothorax did not develop, pneumatoceles developed subsequently in each, while each of the 3 patients without pneumatoceles had a large pyopneumothorax requiring intubation. We have hypothesized that pleural accumulations might have a compressive effect which inhibits the disruption of the small abscess focus.

The only death in the series occurred in a 1-week-old infant with multiple congenital anomalies. An overwhelming infection developed in this case despite adequate pleural drainage. The nature of the pneumonic process is shown in the photomicrograph of the infected lung (see Fig. 1).

All of the remaining patients are now asymptomatic. Five patients have shown x-ray evidence of complete resolution of the pneumatoceles within 1½ to 6½ months after onset of the pneumonia. The remaining 4 patients with pneumatoceles are showing resolution during observation periods of 11 days to 2 months after the onset of the pneumonia.

CASE REPORT

A female infant, aged 2½ months, was transferred to our service from another hospital on Jan. 25, 1956. Although no respiratory difficulty had been demonstrated at delivery and the early neonatal period had been uneventful, nasal obstruction and cough developed on Jan. 5, 1956, when the patient was 53 days old. A roentgenogram of the chest showed opacification of the right lung field. By Jan. 13, 1956, an air-fluid level had developed in the right pleural space. Respiratory distress became increasingly severe. Hemolytic streptococcus and *Staphylococcus aureus* could be cultured from the purulent exudate in this pocket.

On Jan. 14, 1956, myocardial decompensation was suggested by the findings of tachycardia, cardiac enlargement, and hepatomegaly. Digitoxin was added to the current antimicrobial therapy of chloramphenicol and streptomycin, and the right pleural space was drained through the bed of the eighth rib. The infant's condition remained critical following the intubation, and she was transferred to our service.

On admission, the child appeared pale and showed considerable respiratory distress. The heart rate was 164 and respiratory rate was 66. Breath sounds were diminished and the percussion note was flat over the entire right hemithorax. No râles or wheezes were audible;

Fig. 4.—Management of two pneumatoceles by intracavitary suction tube drainage (see case report). *Top Left:* The right lower lung field is replaced by a large tension cyst herniating into the posterior mediastinum. Other pneumatoceles herniate superiorly into the left lung field. When the intrapleural tube was clamped, air leaked into the pleural space, demonstrating the extent of the empyema pocket and the intrapulmonary nature of the cystic shadows. *Top Middle:* After insertion of the tube into the inferior cyst, it was kept deflated but the superior pneumatoceles have expanded. *Top Right:* Lateral view demonstrating deflated posterior pneumatocele and tension cyst anteriorly.

Bottom Left: Both pneumatoceles are deflated by intracavitary suction tubes. The position of the mediastinum was improved and the left lung was better aerated. *Bottom Middle:* When the tubes are clamped for 10 minutes, air accumulates in the upper pneumatocele but not in the lower one. *Bottom Right:* Film 1 year later showing resolution of pleural and intrapulmonary processes.

no stridor could be heard. A No. 14 Fr. catheter was secured in an incision in the right posterior axillary line. The liver was palpable 1.5 cm. beneath the right costal margin. No cyanosis was present.

White blood cells numbered 16,200 per c. mm. with a differential count of 59 neutrophils and 41 lymphocytes. The red cells numbered 3,240,000 per c. mm. Hemoglobin concentration was 9.0 Gm. per 100 c.c. The urine contained white cells and granular casts. A culture of fluid from the thoracotomy tube yielded a heavy growth of beta-hemolytic *Staphylococcus aureus,* which was moderately sensitive to chloramphenicol and bacitracin, slightly sensitive to Achromycin, and resistant to penicillin, streptomycin, Aureomycin, Terramycin, and Aerosporin. An electrocardiogram showed changes consistent with a digitalis effect.

Continuous suction at a subatmospheric pressure of −10 cm. of water was applied to the chest tube. Small amounts of air bubbled from this tube. Digitoxin and transfusions of whole blood were given. Chloramphenicol was administered intramuscularly in doses of up to 200 mg. every 24 hours. Large doses of erythromycin were also given. Heavily-misted oxygen was administered.

On the second hospital day, it was necessary to change the intrapleural tube to establish an air-tight connection through the area of rib resection. The patient then passed numerous loose mucoid stools culturing Pseudomonas organisms. To combat the diarrhea, intravenous fluids were given and neomycin started by gavage to a total of 200 mg. every 24 hours. Although respiratory distress diminished for several days, by January 30 the child was dyspneic and cyanotic. The respiratory rate rose to 60 and was accompanied by soft-tissue retraction. She appeared to be moribund, with signs of vascular collapse. A chest roentgenogram showed that the size of several pneumatoceles had increased as the empyema had resolved. Cysts in the right upper lung field caused the lung to herniate into the superior mediastinum, and a giant inferior pneumatocele herniated into the posterior mediastinum. The entire mediastinum was deviated by the compression. A small amount of air had accumulated within the empyema pocket in the short time required to transport the patient to the Radiology Department. This proved useful, however, in demonstrating the limits of the empyema pocket, excluding pleural loculation as a source of cystlike artifact and delineating the cystic structures as lying within the substance of the right lung.

In view of the critical situation and the possibility that the inferior pneumatocele might be interfering with the circulation by compression of the inferior vena cava, this cyst was intubated. A calibrated 18-gauge needle was inserted into the pneumatocele under Xylocaine anesthesia. As we prepared to measure manometric pressures, the barrel blew out of the syringe. After this incident, the pressure measured +15 to +20 within the cyst and could be reduced to −2 cm. by the withdrawal of about 10 c.c. of air. However, it was possible to continue aspirating unlimited amounts of air, thus confirming the free bronchial passage of air into the cyst. When continuous needle aspiration was interrupted, pressure rose to +20 cm. of water in a matter of 1 or 2 minutes. Respiratory distress was diminished during the moments of needle decompression but increased with interruption of the continuous aspiration. The intrapleural tube showed subatmospheric fluctuations during this period.

In this situation it was obvious that intermittent needle aspiration of the pneumatocele would be ineffective; consequently a catheter was inserted into the cyst by the trocar technique. For 48 hours after intubation large volumes of air continued to bubble, although −15 cm. bottle suction could be maintained. The infant's condition improved strikingly during this period.

Progression of the size of the superior pneumatoceles caused the return of respiratory distress and cyanosis on Feb. 2, 1956. Needle pressures were strongly positive within the superior pneumatocele but remained subatmospheric in the inferior cyst. It was again possible to aspirate large volumes of air only to have it reaccumulate promptly under strong positive pressure. Consequently another tube was inserted into the superior pneumatocele. The following day, roentgenograms were exposed before and after a 10-minute period of clamping of the two pneumatocele tubes. During this time the inferior cyst remained compressed

pneumatocele but not in the lower one. *Bottom Right:* Film 1 year later showing resolution of pleural and intrapulmonary processes.

but the superior pocket ballooned to a large volume. It had been possible to clamp and remove the intrapleural tube without the recurrence of pyopneumothorax despite the fact that the inferior pneumatocele tube, at least, traversed free pleural space.

By February 7, the patient showed steady improvement. No air accumulated within the inferior pneumatocele during a 24-hour period of tube clamping, so the tube was removed. On February 15, the superior cyst tube was removed. During the next few days small amounts of purulent material accumulated within the empyema pocket and were aspirated with a syringe and needle. The tube tracts remained decompressed. Several smaller pneumatoceles in the right lung showed no tendency to expand. Antimicrobial therapy was discontinued on February 24 and the child was discharged from the hospital on February 29 of that leap year. She has remained well to the present date. Roentgenologic resolution of the remaining pneumatoceles occurred within 1 month after discharge, at which time pleural reaction was negligible.

Fig. 5.—Appearance of the child with two intracavitary suction tubes in position.

Comments on Intubation Technique.—Since we have been impressed with the lack of control of the depth of penetration when intercostal tubes are placed by the trocar method, we have used a series of measurements to ensure accurate placement of the tubes both in empyema pockets and in pneumatoceles (which are small in volume compared to the usual tension pneumothorax). The details of placement and measurement are shown in Figs. 6 and 7. Centimeter calibration rings are useful on the Ochsner trocar and 18-gauge aspiration needle, although the same results may be obtained by temporarily marking the necessary measurements on an ordinary trocar.

Rib resection has not been necessary to allow passage of the large trocar through an interspace. Even when the trocar is larger than the space, the mobile rib cage of an infant tends to spread if the blade of the trocar is accurately pointed between the ribs into the intercostal muscle. A flat surface of the triangular trocar blade should be parallel to the edge of the upper rib so as to minimize the hazard of injuring intercostal vessels. The danger may also be lessened by directing the trocar slightly caudalward as the blade penetrates the intercostal bundle. In this manner the trocar point is directed closer

to the upper margin of the inferior rib and away from the intercostal vessels. We have not encountered bleeding after tube placement—a fortunate result of our precautions or of the tamponade effect of the large tubes we have used.

The point of tube passage was selected by careful analysis of posteroanterior and lateral chest roentgenograms. We have felt that fluoroscopy is not necessary and have been reluctant to subject seriously ill infants to the handling which is necessary for such an examination.

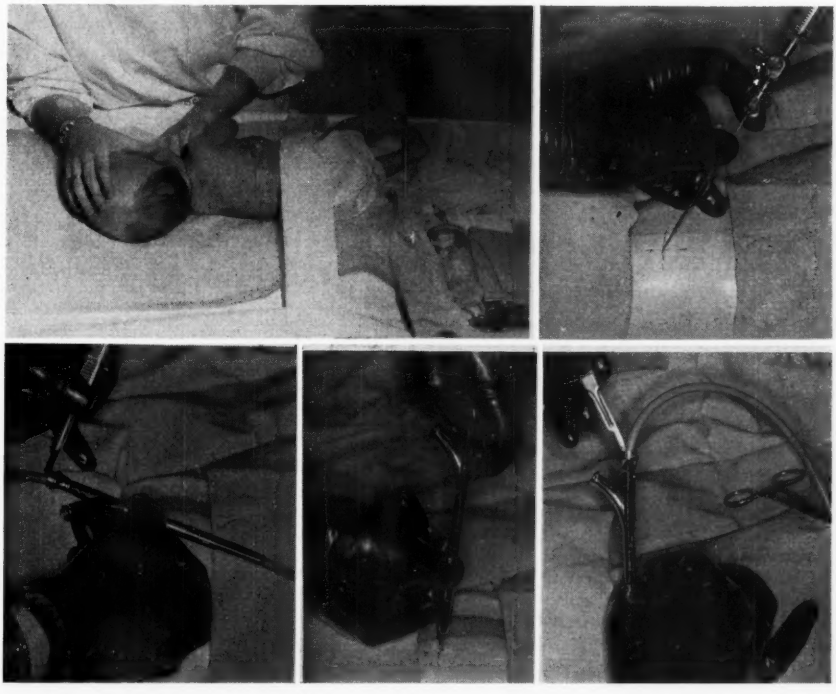

Fig. 6.—Method of insertion of trocar. *Top Left*: Position and restraint of the infant. *Top Right*: Aspiration of the pneumatocele with an 18-gauge needle calibrated at centimeter intervals. Intracystic pressures are measured and the depth required for penetration noted. *Bottom Left*: A catheter is marked in the proper position within the trocar. *Bottom Middle*: The trocar is inserted into the cyst. *Bottom Right*: With the trocar within the cyst, the catheter is adjusted to the correct depth.

Manometric pressure measurements have proved useful in demonstrating the dynamics of gas exchange into the cysts and empyemas. An ordinary pneumothorax machine has been used. The rapid loss of subatmospheric pressure in the minutes after aspiration promptly indicates the necessity for continuous tube drainage. Pressure disparities in various pockets on aspiration indicate the absence of free communication between the various pockets.

The large bore of a No. 16 Fr. catheter is useful in evacuating the large amounts of air and minimizing obstruction caused by grumous staphylococcic

exudate. Both empyema and pneumatocele catheters have been irrigated with small amounts of saline solution to ensure patency. Because of the bronchial communication, we have placed the patient in such a position that the tube is dependent in the thoracic cavity during the period of irrigation. No signs or sequelae have been suggestive of bronchial contamination with use of the irrigating solution.

Since we demonstrated rapid ballooning of one pneumatocele with a 10-minute period of tube clamping, we now transport all patients to the Radiology Department in their beds, with continuous suction drainage being maintained to preclude transient gas accumulations. We do not remove the catheters until it is evident that air-fluid accumulation will not reappear during a 24-hour period of tube clamping.

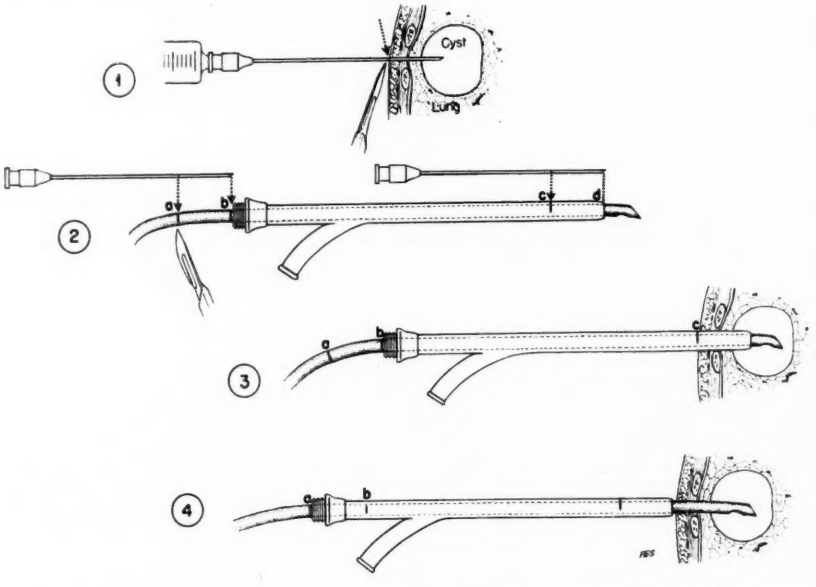

Fig. 7.—Method of adjusting the depth of penetration of the catheter.

(*1*) The cyst is aspirated and the depth of penetration of the needle noted.

(*2*) This depth of penetration is then measured off (*a*—*b*) on the catheter, lying in the correct position in the trocar and on the trocar tip (*c*—*d*).

(*3*) The trocar is inserted to the depth "*c*—*d*," placing its tip within the cavity. The catheter is inserted into the trocar to a depth of point "*b*." It is now in correct position within the cyst.

(*4*) The trocar is drawn back to the skin level and the position of the catheter is adjusted finally by bringing point "*a*" flush with the cannula orifice.

SUMMARY

1. Twelve infants and children with staphylococcic pneumonia were treated at the United States Naval Hospital, Chelsea, Mass., between January, 1956, and April, 1958. Pyopneumothorax developed in 7 of these children. Nine

showed pneumatoceles at some time during the course of the illness. One premature infant died with multiple congenital deformities and pyopneumothorax. The remaining patients are asymptomatic and show roentgenologic changes of resolution.

2. Pyopneumothorax was treated by repeated needle aspirations when slight and by intercostal tube drainage when extensive.

3. All 9 patients with pneumatoceles show either complete resolution within 1½ to 6½ months after onset of the pneumonia, or progressive resolution when observed for periods of less than 2 months.

4. Eight of the patients with pneumatoceles required no specific treatment. In 1 infant, intracavitary suction tube drainage was lifesaving in that it prevented tension distortion of the thoracic contents by two pneumatoceles which appeared early in the toxic phase of the pneumonia. The tubes were introduced without staging the procedure and, in at least one instance, traversed free pleural space without aggravation of the pre-existing empyema. The alternative of cyst excision is regarded as undesirable because of the probability that salvable lung tissue would be excised, and because thoracotomy would be a formidable procedure in the presence of empyema and pneumonic toxicity.

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1. Wallman, I. S., Godfrey, R. C., and Watson, J. R.: Staphylococcal Pneumonia in Infancy, Brit. M. J. 2: 1423, 1955.
2. Rakower, J., and Wayl, P.: Monaldi Drainage in the Management of Postinfectious Pulmonary Cysts, J. Pediat. 52: 573, 1958.

Discussion

(Papers by Maier [page 625] and Watkins [page 642] and Their Associates)

DR. DAVID C. SABISTON, Baltimore, Md.—The rising incidence of all types of staphylococcal infections during the past several years has indeed been impressive. One of the more important problems which we have encountered has been the marked increase of staphylococcic pneumonia occurring in infants and young children. Although once an uncommon disease, it is now of frequent occurrence. I would like to comment briefly on several points regarding management and prognosis in a group of patients studied at the Johns Hopkins Hospital. During the 2-year period, 1951-1952, there was a total of 6 cases of staphylococcic pneumonia seen in the Department of Pediatrics. The striking rise in the incidence of this disease is shown by the statistics of the past 3 years (1955-1958) during which time there have been 61 cases. In this group, 26 or 38 per cent have been managed by closed intercostal catheter drainage in addition to antibiotic therapy.

Staphylococcal Pneumonia in Infancy and Childhood—61 Cases

Closed intercostal drainage (catheter)			23 (38%)
Open drainage			1
Pulmonary decortication			1

Results in Staphylococcal Pneumonia, 1955-1958

	Cases	*Deaths*	*%*
Primary	55	4	7
Secondary*	6	6	100
Total	61	10	17

*Primary disease: cystic fibrosis, nephrosis, hypogammaglobinemia, and postoperative.

(slide) The indications for drainage have been primarily the presence of large tension pneumatoceles, a tension pneumothorax, or extensive empyema. Of the 61 patients studied, 55 were primary and in this group there were 4 deaths (7 per cent).

(slide) It is interesting that 3 of the 4 patients who died were less than 3 months of age and, in each instance, the course was characterized by a rapidly progressive toxemia leading to death within 48 hours. Each of the 6 patients who had staphylococcic pneumonia secondary to a systemic disease died as a result of the combination of their illnesses.

We agree fully with the authors that catheter drainage should be employed frequently in the management of the intrathoracic tension complications which are often associated with staphylococcic pneumonia in children. It has been our experience that it is often necessary to place multiple catheters within the chest and that, in some instances, these must be replaced on several different occasions. All the patients we have observed have been treated with a combination of large doses of bacteriocidal and bacteriostatic drugs. Penicillin, in combination with either Chloromycetin or Albamycin, has been the antibiotic of choice in the majority of cases. With the combination of chemotherapy and closed surgical drainage, good results have been obtained in the patients with primary disease. Open drainage has been necessary in only one instance.

In the last slide, the initial chest film of this patient demonstrates multiple small pneumatoceles and empyema in the right lung. A tension pneumothorax developed and this failed to respond to an intercostal catheter. Later it was necessary to treat the child with rib resection and open drainage. Marked clinical improvement followed but the second film shows a large empyema cavity demonstrated by the injection of contrast media. Pulmonary decortication was then performed by the Resident, Dr. Merril Brown, with complete re-expansion of the lung. The final film shows the late postoperative result and the child is now well.

In closing, I should like to compliment Dr. Watkins and Dr. Hering on their very interesting presentation.

DR. JEROME R. HEAD, Chicago, Ill.—In reading the abstract of this paper in the program, I was very much interested to learn how a catheter could be put into an intrapulmonary cavity with trocar and cannula without the production of pneumothorax. Now, having heard the paper, I can understand that this was attempted and that it was successful because the patient had had an empyema which ensured the presence of adhesions between the lung and the chest wall. My experience with intracavitary suction has been limited to tuberculosis and large emphysematous blebs and in these cases it is essential to do a two-stage operation or, at least, to be sure that the two layers of pleura are adherent before making an opening into the lung. I think that it would be too bad if anyone left this meeting with the feeling that under ordinary circumstances it is safe to insert a trocar into the lung without first exploring the pleura.

DR. MAX G. CARTER, New Haven, Conn.—May I congratulate Dr. Watkins for an excellent presentation of an increasingly important subject. I would like to re-emphasize what has been stated about the necessity to distinguish between the presence of a true pleural empyema and intrapulmonary air cysts arising as a result of staphylococcal infection. We see 10 or more of these patients each winter and have observed the development of cystic areas in the involved lung which may persist long after the temperature has become normal and the patient looks clinically well. Ultimately, within 3 to 6 months these all clear.

On the other hand, an actual empyema demands prompt attention by intercostal catheter as discussed by Dr. Watkins. It has been our practice to use a small polyethylene catheter placed through an intercostal needle early in the disease when the fluid is thin, thereby achieving total expansion with little trauma. If thick purulent material is present, of course a larger catheter is needed.

DR. OSLER ABBOTT, Emory University, Ga.—This discussion was not returned.—EDITOR.

DR. WATKINS (Closing).—I want to thank all the discussers for their comments. We had hoped to promote such an appraisal of the current status of staphylococcic pneumonia. In relation to Dr. Potts's comments, we have been impressed with the fact that patients developing pneumatoceles have all had very large pyopneumothoraces, whereas there seems to be an increased incidence of pneumatocele formation in patients who do not develop pyopneumothorax. We have postulated that the compressive effect of the pyopneumothorax may inhibit disruption of the small staphylococcic abscesses which give rise to the pneumatoceles. This reported case differed from the usual patient only in that pneumatoceles developed during a period when the pleural process was active and the patient was toxic. Otherwise, pleural and cyst complications appeared typical.

I would comment on Dr. Head's remarks by stating that I would not hesitate to intubate across a free pleural space if the situation warranted because of the serious nature of the disease and the urgent necessity for definitive correction. I would attempt to obtain artificial pleural symphysis using the method described by Dr. Abbott, but even if this failed, it would actually seem best to intubate directly across a free pleural space (under antibiotic therapy) and watch carefully for a pleural leak requiring pleural intubation. The only alternatives to this course of action are to do a thoracotomy, certainly lethal, waste valuable time staging a Monaldi type of intubation, or allow an intolerable tension situation to continue. Fortunately, I suspect that we will only see trouble from tension pneumatoceles when the patient is ill from pleural complications.

We have made every effort to use large tubes because of the large volume of the air leaks and because of the nature of the exudate aspirated from a pneumatocele. The exudate, which is usually small in volume, is very thick and tends to occlude small tubes. Consequently, we have used No. 16 Fr. catheters to introduce into the cysts. I would not use a plastic tube passed through a needle because it has an exceedingly small lumen, and I would think that it would become blocked rapidly.

TOPOGRAPHY OF THE HUMAN CORONARY ARTERIES IN RELATION TO CARDIAC SURGERY

Thomas N. James, M.D. (by invitation), and George E. Burch, M.D. (by invitation), New Orleans, La.

DISTURBANCES in cardiac rhythm and conduction of impulses are recognized as important problems in cardiac surgery. Although every attempt is made to spare the conduction pathways and the sino-auricular (SA) and atrioventricular (AV) nodes, little attention has been directed to the blood vessels supplying these areas. This has been largely due to meager knowledge regarding their circulation.

In a study of 70 human hearts by injection of Vinylite into the coronary arteries, followed by hydrochloric acid corrosion, observations have been made on the blood supply to the conduction system. The study provided a spatially oriented preparation of the heart chambers and vessels.

Detailed descriptions of the blood supply of the human atria and of the interventricular septum have been published.[1, 2] These regions embrace all but the terminal portion of the human cardiac conduction system.

To demonstrate how blood supply to conduction centers may be disturbed during cardiac surgery, four surgical procedures in current use will be discussed. These particular procedures were chosen because they involve different areas of the heart and, by understanding coronary topography in relation to them, one can anticipate the problems of other surgical procedures in the same areas.

I. THE PURSE-STRING REPAIR FOR MITRAL INSUFFICIENCY[3, 4, 5]

Anteriorly this purse-string suture is tunnelled beneath the left circumflex coronary artery toward the anterior interatrial sulcus, then is inserted into the right atrium. As the suture enters the right atrium, the blood supply to the SA node may be jeopardized in the following fashion. The artery to the SA node (ramus ostii cavae superioris) arises from the proximal left coronary artery (Fig. 1) in 40 per cent of cases and from the right coronary artery (Fig. 2) in 60 per cent. From either origin, this vessel passes behind the aorta

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Read at the Thirty-eighth Annual Meeting of The American Association for Thoracic Surgery at Boston, Mass., May 16-18, 1958.

Fig. 1.

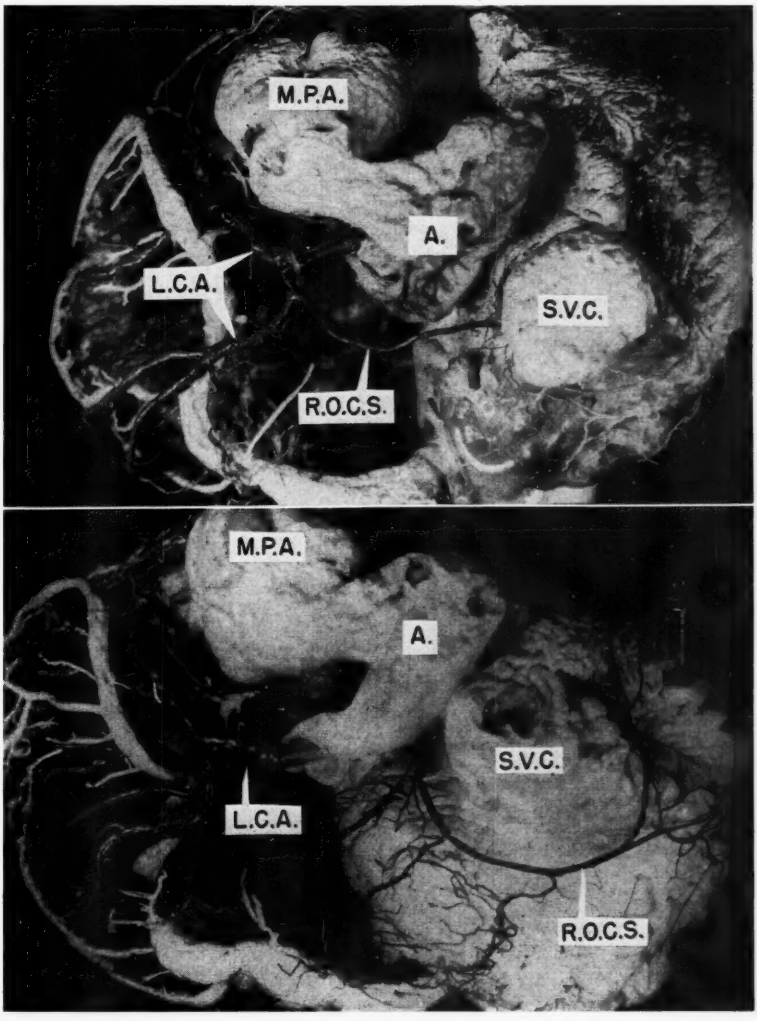

Fig. 2.

Fig. 1.—Photograph of a cast from a heart in which the ramus ostii cavae superioris (*ROCS*) arises from the left coronary artery (occurs in 40 per cent). White plastic casts the right atrium, the superior vena cava (*SVC*), the main pulmonary artery (*MPA*), and the coronary sinus with the venous branches. There is also white plastic in the root of the aorta (*A*) and a bridge of plastic fixing the aortic root to the main pulmonary artery. *LCA* is the left coronary artery. The left atrium and ventricle are not cast. Note how the ramus ostii cavae superioris crosses the region of the interatrial septum anteriorly and encircles the base of the superior vena cava, where it branches to supply the SA node.

Fig. 2.—Photograph of a cast from a heart in which the ramus ostii cavae superioris arises from the right coronary artery (occurs in 60 per cent). Casting and identifications are the same as in Fig. 1.

to the anterior interatrial septal groove, ascends to the base of the superior vena cava and encircles this base to supply the SA node.[1] When the suture is passed into the right atrium it may encircle the SA nodal artery, particularly when it arises from the right coronary artery (60 per cent of our cases). As the suture is tightened, the blood supply to the SA node may be occluded. Consequences will depend on collateral supply available to the node, but may include sinus arrest, atrial fibrillation, or other atrial arrhythmias.

Posteriorly, the suture is passed out of the right atrium beneath the coronary sinus to the posterior surface of the left ventricle just below the AV sulcus. There are two hazards at this point. First, the artery to the AV node (ramus septi fibrosi) penetrates the junction of the interatrial and interventricular septa at this location, arising from whichever coronary artery passes the crux of the heart; in 85 per cent of our cases this is the right coronary (Fig. 3). Since the purse string is placed around this artery (Fig. 4), tightening of the suture may occlude the vessel supplying the AV node. The consequence of this occlusion again depends on collateral supply available, but disturbances in AV conduction such as complete or incomplete block may develop.

The second danger at this location (the crux) is to the posterior descending coronary artery, which frequently makes a deep U-turn beneath the posterior interventricular vein just before it descends in the interventricular groove (Figs. 3 and 4). At the apex of this U-turn the AV node artery usually arises. In addition to the AV node artery, this loop of the posterior descending coronary artery which is not visible on the surface may be occluded when the purse-string suture is passed beneath the coronry sinus. Consequences of such an occlusion may be posterior myocardial infarction, the size governed by the extent of collateral circulation available.

Completion of the purse-string suture anteriorly is guided by the location of the straight branches of the left circumflex coronary artery, with the suture being passed beneath each of these, most of which are visible in the epicardium.

II. THE CIRCUMCLUSION TECHNIQUE FOR CLOSURE OF INTERATRIAL SEPTAL DEFECTS[6, 7, 8]

In this operation, a suture is passed within the base of the interatrial septum, entering the septum by passing above the right coronary artery and behind the aorta and emerging posteriorly above the coronary sinus (Fig. 5). As a supplementary maneuver, the top of the interatrial septum is divided as deeply as possible in a sort of cleavage into the septum for the purpose of freeing the neighboring superior vena cava and medial pulmonary veins from traction when the circumclusion suture is brought together over the top of the heart and tightened. In his description of this cleavage of the upper septum, Sondergaard[8] calls attention to a small vessel just in front of the right medial pulmonary vein which must be severed, but states that there is otherwise no bleeding.

Some of the problems of the circumclusion procedure in regard to the coronary arteries are apparent from the previous discussion of the mitral purse string. The cleavage of the upper interatrial septum will almost certainly

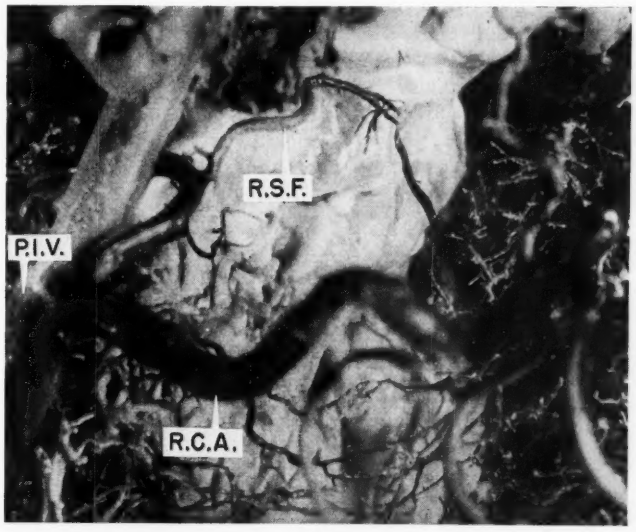

Fig. 3.—Photograph of a cast of the right coronary artery, its U-turn, and the branch to the AV node. White material in the background is a cast of the left atrium (*above*) and the left ventricle (*below*). The right coronary (*RCA*) curves in the foreground then makes a U-turn deep to the posterior interventricular vein (*PIV*), which is cannulated. At the apex of the right coronary loop the branch to the AV node, ramus septi fibrosi (*RSF*), arises.

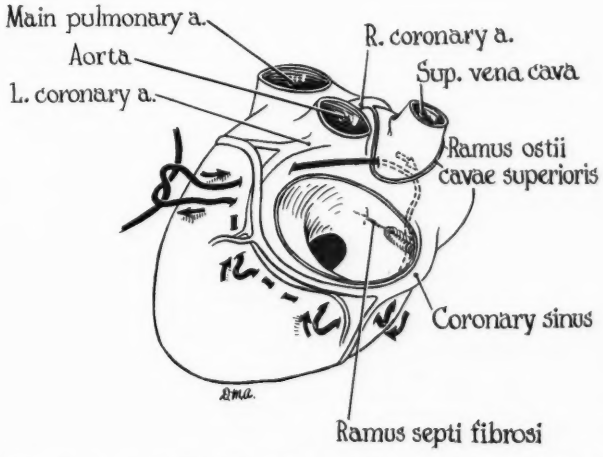

Fig. 4.—Diagram of the mitral purse-string operation for repair of mitral insufficiency. Note how the suture may damage the arteries to the SA node (ramus ostii cavae superioris) or the AV node (ramus septi fibrosi). See text for discussion.

compromise the blood supply to the SA node (Figs. 1, 2, and 5) and, indeed, the vessel which Sondergaard calls attention to as a bleeding hazard is very likely the ramus ostii cavae superioris which supplies that node. The artery to the SA node also is endangered anteriorly when the circumclusion suture is passed into the base of the interatrial septum.

The artery to the AV node and the upper course of the posterior descending coronary artery (Figs. 3 and 5) are jeopardized posteriorly, both by the passage of the suture in the base of the interatrial septum and by traction when the circumclusion is completed.

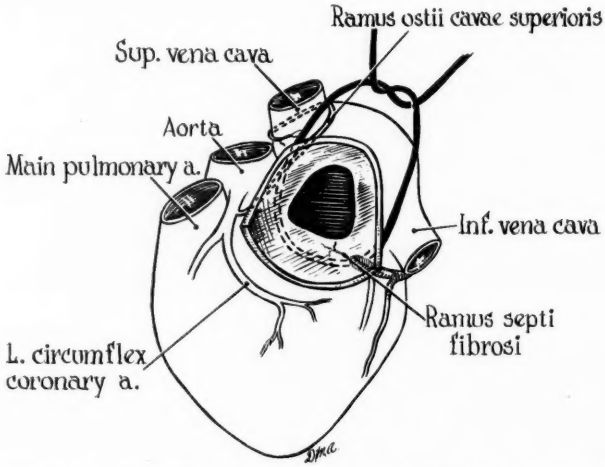

Fig. 5.—Diagram of the circumclusion operation for closure of interatrial septal defects. The arteries to both the SA and AV nodes may be jeopardized (discussed in text).

In addition to the arteries supplying the two nodes, circumclusion encounters a unique bleeding hazard in the lower interatrial septum. The loose areolar connective tissue in this region, through which probe dissection is physically simple, is full of thin-walled Thebesian veins and sinuses. These communicate with the interior of the right atrium.[1] They may be disrupted as the suture is placed, and even more widely opened when it is tightened for the purpose of circumclusion.

III. CANNULATION OF THE VENAE CAVAE FOR TOTAL CARDIAC BYPASS

These cannulae are usually inserted through the atrial appendage or the lateral wall of the right atrium and passed into the venae cavae, where they are secured by tapes (Fig. 6). The blood supply to the SA node may be disturbed by the tape securing the superior vena caval cannula if it is placed near the base of the vena cava, where the arterial circle supplying the SA node lies (Fig. 7).

IV. RIGHT VENTRICULOTOMY

The upper end of this incision is usually near the level of the pulmonic valve ring (Fig. 8). At this point there is an arterial circle (Vieussens' ring)

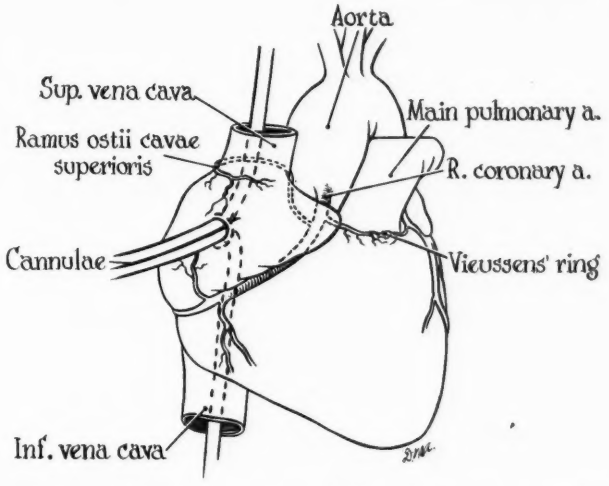

Fig. 6.—Diagram of transatrial caval cannulation, such as may be employed in cardiac bypass procedures. The purse-string incision in the atrial wall may disrupt one of the major channels of collateral circulation to the SA node (from the right intermediate atrial artery). More important, the tape to secure the superior vena caval cannula may be tightened around the ramus ostii cavae superioris arterial circle which supplies the SA node.

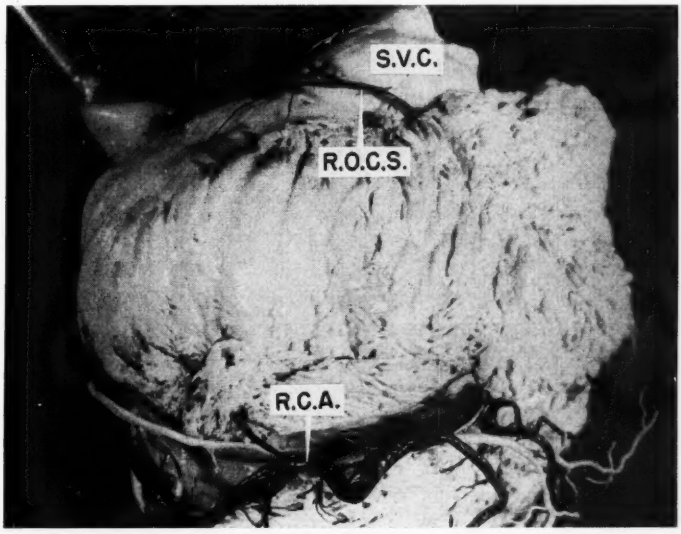

Fig. 7.—Photograph of the lateral aspect of a cast of the right atrium, viewed from just above the margo acutus. Note how the location of the arterial circle of the ramus ostii cavae superioris (*ROCS*) about the base of the superior vena cava (*SVC*) exposes it to the procedure diagrammed in Fig. 6. The right coronary artery (*RCA*) emerges from beneath the right atrial appendage in the right half of the photograph.

connecting the proximal left and right coronary arteries. It is easily identified in the beating heart and whenever possible should be spared because of its importance as a collateral source of flow to either main coronary artery.

The interventricular septum normally lies quite near the free wall of the right ventricular outflow tract (Fig. 8). The arteries of the interventricular septum are predominantly derived from the left anterior descending coronary artery and course posteriorly in a superficial location on the right side of the septum, lying virtually in the right ventricular endocardium.[2] Cutting these arteries in the anterior third of their course may compromise the supply of the distal septal myocardium, as well as the bundle branches of the conduction system.

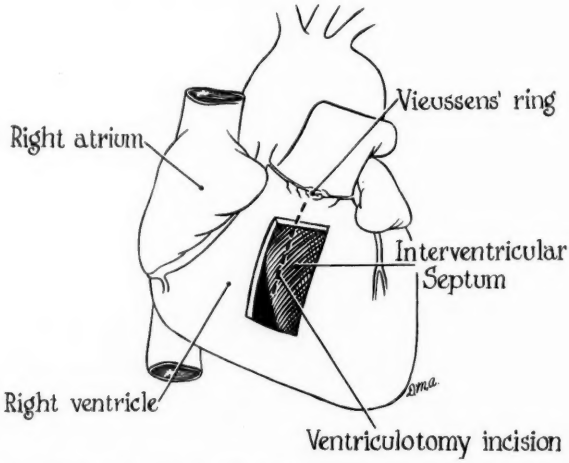

Fig. 8.—Diagram of the incision for right ventriculotomy. The incision should begin below the ring of Vieussens (see text). A window is illustrated in the wall of the right ventricle to expose the underlying interventricular septum, which is in close proximity to the free wall in the region of the pulmonary conus.

DISCUSSION

Consideration of the anatomy of the human coronary arteries suggests that some of the arrhythmias and conduction disturbances encountered in cardiac surgery may be due to impairment of the blood supply of the major conduction centers rather than to damage of the conducting tissue directly. In particular, this may apply to the arteries supplying the SA and AV nodes.

Description of the above hazards is not intended to detract from the surgical procedures discussed, since their merits must be evaluated on many other considerations. It is rather to be hoped that by describing these particular problems all cardiac surgical procedures may be made safer.

SUMMARY

Topography of the human coronary arteries has been discussed by considering it in relation to four surgical procedures in current use. Some of the

arrhythmias and conduction disturbances encountered in these and other operations on the heart may be the result of impairing the blood supply to conduction centers.

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DISCUSSION

DR. PAUL DE CAMP, New Orleans, La.—I want to congratulate Dr. James on this beautiful anatomic study which he has made, and on his careful correlation of it with surgical problems. It is perhaps a commentary on current surgical practice that the surgeons should be so busy operating on beasts and man, so busy making speeches and writing papers that it is necessary for an internist to make such an important anatomic study.

We feel that it is an important concept that cardiac irregularities following operations on or within the heart may be due to interference with the vascular supply to the neuromuscular mechanism, rather than necessarily due to direct trauma to the nervous mechanism itself. It would, of course, be characteristic of vascular involvement that, in some cases, it would be reversible as collateral circulation builds up to compensate for the initial vascular injury. That, of course, is frequent in clinical experience. However, we would like to report one case in which apparently permanent disabling cardiac irregularities developed following an operation in which we suspect the injury to the neuromuscular mechanism was due to involvement of the blood supply. This is the case of a 31-year-old man with a large interatrial septal defect. In March of 1956 this defect was closed by the Sondergaard type of purse-string ligature. This purse string was passed through the interatrial supraventricular tunnel and at no time passed through the myocardium itself, so that injury to the A-V node or bundle of His seems extremely unlikely. Beginning promptly, following the operation and continuing to the present, the patient has had a series of disabling mechanical disorders, including auricular fibrillation, supraventricular tachycardia, probably a flutter, auricular flutter with a shifting pacemaker, auricular fibrillation, auricular tachycardia, auricular flutter, and presence of a third pacemaker. These slides demonstrate some of the electrocardiographic abnormalities which have been noted during this period. With vigorous medical management we have been able to control the rate fairly well, but this patient has never been able to go back to work since his operation.

DR. J. C. DAVILA, Philadelphia, Pa.—We have been aware of the fine work of Dr. Burch and Dr. James since their recent publication, and have naturally been most interested in their findings and their description of the coronary blood supply to the atria and to the

S-A node. It is not unlikely that some of these vessels may be injured by some of the procedures performed in these areas. However, the crux of the matter lies in the results which are obtained in these operations, realizing that there are certain structures which, in all types of surgery, must be sacrificed, provided this does not result in serious consequences.

With regard to the mitral purse-string operation, we have seen in our series 8 patients with postoperative atrioventricular dissociation, 2 of which had preoperative suggestion of partial A-V block. All but one of these 8 patients have reverted to their preoperative rhythm within the first 2 to 4 weeks following surgery. Whether this incidence of conduction defect can be attributed to injury to the blood supply of the S-A node by the surgical operation is certainly a moot question.

In some 20 cases of interatrial septal defect of the ostium secundum type, in which we have operated by the Sondergaard method, we have not seen any arrhythmias which we would attribute to surgery. In one patient there was paroxysmal ventricular tachycardia preoperatively, manifesting itself in frequent short bursts of ventricular activity. Following surgery, this patient's episodes of ventricular tachycardia became no less frequent but tended to persist a little longer. After several such bouts within the first 48 hours following surgery, the patient died when the arrhythmia persisted longer than was tolerable. It was noted at surgery in this patient that we were dealing with an extremely irritable myocardium. Whether the operation performed aggravated the arrhythmia is again a question which we cannot answer.

I should like to congratulate the authors for this excellent presentation.

DR. JAMES (Closing).—I thank the discussers for their interest and comments.

OPEN-HEART SURGERY FOR MITRAL INSUFFICIENCY

Donald B. Effler, M.D., Laurence K. Groves, M.D., William V. Martinez, M.D. (by invitation), and Willem J. Kolff, M.D. (by invitation), Cleveland, Ohio

THE NEED for a direct approach to the mitral valve has long been appreciated, and now the advent of open-heart surgical techniques that utilize the pump-oxygenator has made possible a direct approach to the left atrium and the mitral valve. Constant improvement in Lillehei's[1] original techniques utilizing cross-circulation, and the addition of valuable adjuncts, permit unhurried operations within each chamber of the heart. Although the ultimate in the treatment of mitral insufficiency has not been accomplished, it is possible to provide surgical palliation in the majority of patients.

Our interest in mitral insufficiency dates from early attempts to close interatrial septal defects of the ostium primum type. At that time we did not understand the significance of associated mitral insufficiency and, as a result, in four consecutive operations where the defect was completely and successfully closed, left heart failure occurred. In each case, the technical success was accompanied with worsening of the condition, and 3 of the patients ultimately died of cardiac disease. A similar penalty was attached to our first effort to correct congenital mitral regurgitation by direct suture of the thin leaflets: the initial surgical success was destroyed by the sudden fenestration and partial avulsion of the septal leaflet. This painful lesson impressed upon us the important kinetic difference between operations involving the right and the left ventricle. Time, and additional experience, have brought into better focus the over-all problems related to mitral incompetency. It is our belief that most types of mitral insufficiency may be helped by the open approach that utilizes the pump-oxygenator. The anticipated degree of success will be directly related to the type of valvular defect and the concomitant myocardial and pulmonary changes that are present.

EXPERIENCE

At this time, utilizing the open-heart technique, 14 patients have been operated upon for correction of mitral insufficiency. As mentioned before, at

From the Department of Thoracic Surgery and the Department of Artificial Organs, The Cleveland Clinic Foundation, and The Frank E. Bunts Educational Institute, Cleveland, Ohio.

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least 4 patients were operated on for correction of septal defects without regard for their coexisting mitral insufficiency; these patients are not included in the above group in whom correction of the mitral valve was attempted.

Incompetency of the mitral valve is produced by a variety of pathologic circumstances that we have grouped according to our findings at the time of surgery.

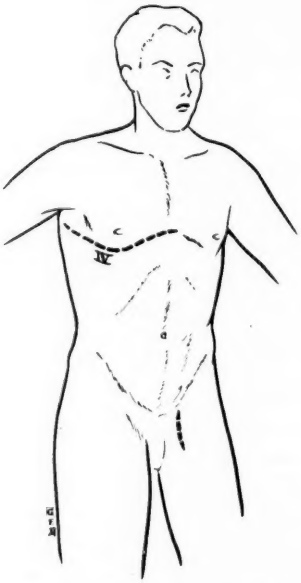

Fig. 1.—Schematic drawing of the right-sided approach to the mitral valve. The anterolateral thoracotomy incision is carried through the right fourth interspace. The sternum will be transsected after division of both internal mammary bundles; the left pleural space will not be entered.

The left common femoral artery is the usual site of retrograde cannulation for arterial return from the pump-oxygenator. The femoral artery is reconstructed after decannulation while the patient is still heparinized.

A. Congenital mitral insufficiency.
B. Acquired mitral insufficiency.
   a. Ruptured chordae tendineae.
   b. Dilatation of the annulus.
   c. Destruction or distortion of the leaflet.
   d. Iatrogenic insufficiency (surgical laceration).

At this stage of operative development, the etiopathogenesis of mitral regurgitation is of less practical importance than are the topographic aspects of the valve itself. Since valvular replacement is not yet feasible, any amelioration of the disease will depend upon the surgeon's ability to make use of what he finds at the site of operation. Hence, the type of regurgitation present, and the anatomic features will have a direct bearing on the surgical outcome.

Every surgeon who employs the routine closed approach for the correction of mitral stenosis will recall the occasional patient in whom was encountered a

greater extent of mitral insufficiency than he had anticipated. Now, in such a circumstance, it is the responsibility of the surgeon to perform meticulous digital examination of the valve with predominant insufficiency, in order to evaluate it for a possible direct, open approach.

TECHNIQUE

Approach.—It is our belief that the surgical approach to the mitral valve is best accomplished from the right side (Fig. 1). As an elective procedure, the

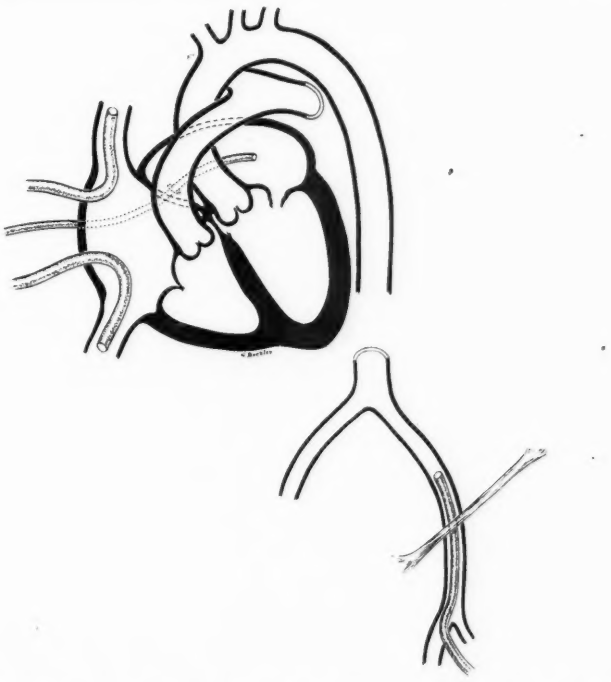

Fig. 2.—Cross-section diagram to illustrate the two approaches to the mitral valve from the right pleural space. Both routes are available through a right thoracotomy incision.

By incising the posterior interatrial groove the left atrium is entered; however, the valvular annulus is not on a direct plane with this portal of entry. A more direct approach is obtained by entering the right atrium first and then incising the interatrial septum within the foramen ovalis.

approach from the right permits a unilateral thoracotomy when retrograde femoral cannulation is employed. The advantages of unilateral thoracotomy are self-apparent: retrograde femoral cannulation is an effective method of rendering arterial return from the pump-oxygenator and, in some respects, is superior to the route through the subclavian artery. The actual entry into the left atrium may be accomplished in either of two ways: (1) through the fossa ovalis by way of the right atrium, and (2) through the posterior interatrial

groove. There are distinct advantages to each mode of entry. The trans-right-atrial approach offers more direct visualization of the medial commissure, reduces the hazard of postoperative bleeding, permits easier placement of sutures, and allows better control of return to the coronary sinus. The approach via the interatrial groove eliminates entry into the right heart and its attendant problems.

We have successfully performed 3 operations through the left-side approach utilizing a transsternal, bilateral incision with entry through the left atrial appendix. In each case, it was necessary to use elective cardiac arrest in order to rotate the large heart from its normal position. The exposure was fairly adequate, but considerable retraction was necessary before the sutures could be properly placed. It was feared that contained air would be a problem, but it was not. However, the closure of the left auriculotomy, and the replacement of the heart into the normal position put the incisional repair in a posterior position where it was difficult to inspect it for residual bleeding. Although the direct left approach is feasible, even preferable in selected cases, it is our belief that access to the mitral valve from the right side has greater advantages. The left approach is probably preferable for myxoma of the left atrium.

Elective Cardiac Arrest.—Induced cardiac asystole is an accepted adjunct to open-heart surgery.[2-6] Our great enthusiasm for the Melrose technique has been expressed in previous reports. Nevertheless, we are not convinced that it always is necessary or advisable to stop the heart before attempting to correct the incompetent mitral valve. As mentioned by Lillehei,[7] there will be occasions when direct visualization in the beating heart may be particularly helpful in evaluating a diseased mitral valve. It is conceivable that the surgeon may create serious distortion when operating on an arrested flaccid heart, particularly when the repair is confined to the annulus. Although our early efforts were all made in the arrested heart, an effort now is made to work initially with the decompressed, beating heart. If, however, the aortic valve is not competent, the return flow to the open heart will be of such magnitude that cardiac arrest becomes necessary. Usually this can be determined after the patient has been put on total bypass—if the heart visibly becomes decompressed, and palpable pressures decrease accordingly, then asystole will not be necessary. On the other hand, should the heart show little reduction in size and the pressures remain elevated, then cardiac arrest is employed and the quiescent heart is decompressed by direct aspiration.

Left Heart Decompression.—A more recent adjunct to open-heart surgery is the use of left heart decompression by an indwelling catheter in the left atrium. This cannula is inserted from the right side through a stab wound in the right superior pulmonary vein as it enters the left atrium (Fig. 3). This simple addition to the routine system of perfusion cannulation permits a constant pressure recording of the left atrial chamber—more important, it permits prompt venting of the blood should there be a dangerous rise in left heart pressure. Muller, Littlefield, and Dammann[8] have reported on the pulmonary changes attributable

to high retrograde pressure in experimental procedures. In a recent report, Kolff and associates[9] recounted our clinical observations in patients who sustained pulmonary damage from high left heart pressures that were transmitted to the pulmonary vascular system. This pulmonary vascular damage is characterized by interstitial hemorrhage, and, on gross examination, is not unlike the picture of blast or compression injury of the lung.

A double purpose is served by the indwelling cannula in the left heart; it decompresses the left heart, and it also permits withdrawal of air from the left atrium. Since trapped air presents an obvious hazard, this additional feature augments the value of this simple safeguard.

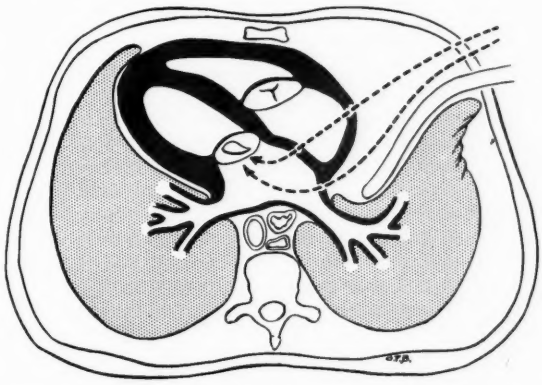

Fig. 3.—Schema of cannulations employed in the open approach to the mitral valve. Three venous cannulae are inserted from the right thorax; 2 cannulae drain the venae cavae in conventional fashion, the third is inserted into the left atrium via the base of the right superior pulmonary vein. This left heart cannula may be used to decompress the left heart, remove trapped air, and measure left atrial pressures.

Retrograde arterial perfusion permits a single thoracotomy approach. The common femoral artery is exposed and, after heparinization, the cannula is inserted through a small vertical incision. After decannulation, the arteriotomy closure is performed while the patient is still heparinized.

Operation.—The patient is prepared for the conventional cardiotomy that utilizes total bypass perfusion. At the Cleveland Clinic Hospital, a disk-type of oxygenator (Kay-Cross* modification of the Björk) is utilized with the roller type of pumps. By direct cutdown the right femoral artery and veins are cannulated for continuous reading of arterial and venous pressures. The electrocardiogram and electroencephalogram are continuously monitored.†

The patient remains in a supine position with the right side of the chest slightly elevated. The incision for anterolateral thoracotomy is made through the fourth interspace, the sternum is transsected, and both internal mammary bundles are divided and are ligated. The left pleural space is not entered deliberately. The pericardium is opened widely and pressures are measured in the chambers and great vessels. Both venae cavae are mobilized, looped by umbilical tape ligatures, and cannulated after heparinization. The left common

*Manufactured by Pemco, Inc., Cleveland, Ohio.

†With the Monitorscope, manufactured by Essential Electronics Corporation, Cleveland, Ohio.

femoral artery is exposed and is cannulated in a retrograde fashion for the arterial return from the pump-oxygenator. A third cannula is inserted into the left atrium as has been described above. The ascending aorta may be mobilized should elective cardiac arrest be necessary.

When high flow perfusion is established and the caval ligatures are secured, the heart will become decompressed and pressures in both sides will fall to negligible levels. If the heart does not become decompressed satisfactorily, and

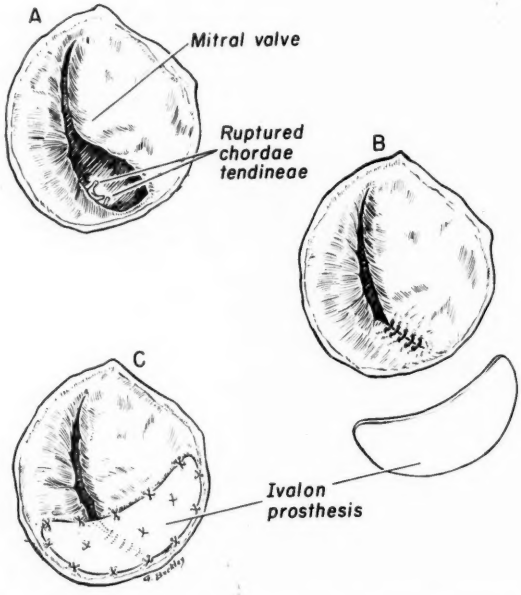

Fig. 4.—Method of correction for flail valve as seen in ruptured chordae tendineae. This method may have special application in iatrogenic insufficiency when chordae tendineae have been surgically transsected.

No effort is made to reapproximate the severed chordae tendineae. The leaflets are approximated over the involved area by direct suture. The valve is then buttressed from the atrial side by a crescent-shaped Ivalon prosthesis that is sutured in place, first to the annulus and then to the underlying valve leaflets. Failure to utilize this reinforcement may be followed by fenestration of the mitral leaflets.

the left heart pressures remain high, it is to be assumed that the aortic valve is incompetent and that elective cardiac arrest should be employed. When aortic insufficiency is not present, there should be no blood return to the heart except that through the coronary sinus and the bronchial vessels.

The right atrium is then opened widely and the coronary sinus return is removed by continuous low pressure aspiration; this blood is diverted to the venous side of the pump-oxygenator. Entry to the left atrium is then obtained by an incision in the fossa ovalis—the exposure is adequate and there is little danger of injuring conduction pathways. Blood is aspirated from the left atrium, and the mitral valve is examined under direct vision. The method of

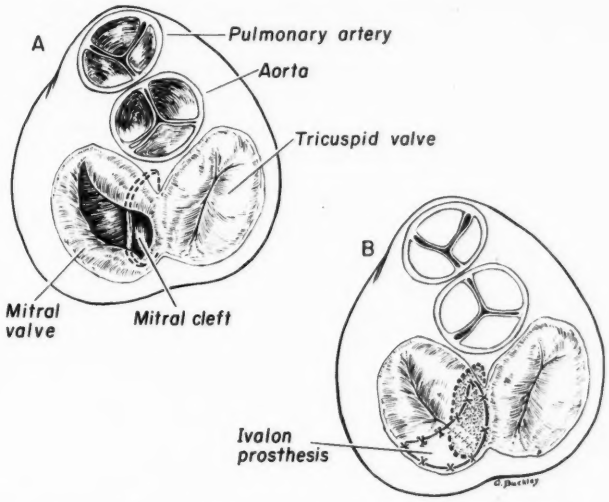

Fig. 5.—Diagram of the approach to the mitral valve through the right atrium. In patients with congenital mitral insufficiency related to interatrial septal defects, the right atrial approach permits adequate correction of the incompetent medial commissure, which may be reinforced as described in Fig. 4. The septal defect is then closed with or without prosthetic appliance.

Application of this technique to acquired mitral insufficiency by creating a temporary septal defect in the fossa ovalis has been rewarding. The right-sided approach through the right atrium and the fossa ovalis appears to be the most direct plane to the mitral valve. Working through the artificial atrial septal defect permits direct visualization of the entire mitral annulus and both commissures.

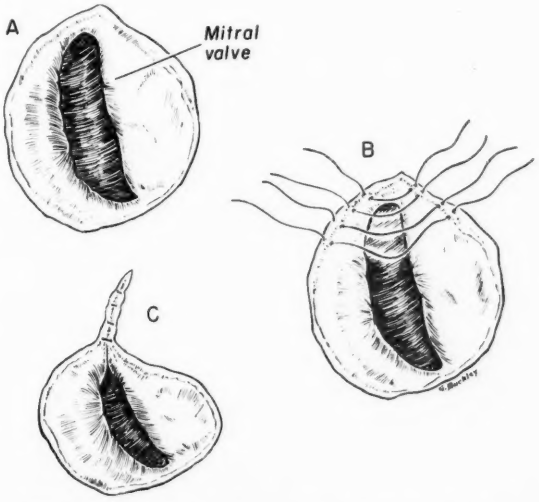

Fig. 6.—A significant number of patients with mitral insufficiency demonstrate only dilatation of the annulus. These patients may not show any stigmata of rheumatic fever. At the present time it would appear that application of the annulus over the anterolateral commissure would offer the best palliation. The valve leaflets in such cases are thin and easily torn; for that reason, no effort is made to close a commissure by direct suture of the leaflet. At the present time, this plication is accomplished by a series of carefully placed silk sutures without prosthetic support.

repair is determined after careful inspection of the diseased valve. If any element of stenosis is present, it may be relieved by scissor or knife incision in the involved commissure. Relief of the mitral insufficiency is then undertaken by direct suture of the leaflet or the annulus, with or without a prothesis. The surgeon may work without hurry and with good exposure of the operative field. Although the left atrium is filled with air, the ventricle is dependent in this position and air embolization during the open procedure has not occurred.

Upon completion of the mitral valvuloplasty, the left atrium is allowed to fill with blood before the septal defect is closed by continuous suture. At this

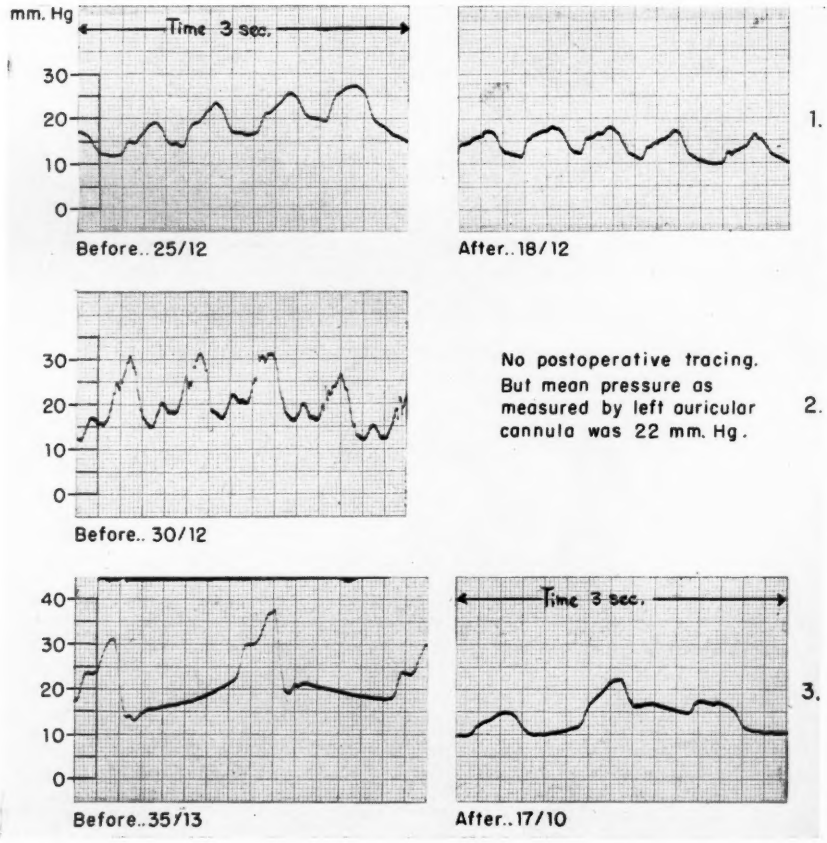

Fig. 7.—Pressure recordings on the operating table before and after attempts to correct mitral insufficiency on 6 consecutive patients. (These pressures were recorded by Miss Rose Litturi with a Sanborn strain gauge amplifier, Model No. 140, Serial No. 440, and Statham pressure transducer, Model P23D, 1-75 cm. Hg, Serial No. 295.)

Case 1.—Left atrial pressure was reduced.

Case 2.—This patient had iatrogenic split of one leaflet of the mitral valve plus severance of the chordae tendineae. After the first attempt at open surgical correction, mean pressures in the left atrium were found of 33 mm. Hg. After a second attempt, these fell to 22 mm. Hg. The surgical problem could not be solved.

Case 3.—Left atrial pressures were considerably reduced and the right ventricular pressure was also reduced from 36/2 to 20/7 mm. Hg.

time the indwelling cannula in the left heart is vented with low negative pressure to remove any residual air trapped in it. The right atrial incision is then closed, and the caval ligatures are released while the heart is being filled from the right side.

It is our practice to continue the perfusion for 10 minutes or more after the heart has refilled and its beat has returned to a satisfactory rhythm. Since it is possible for a small amount of air to be trapped in the left heart for a number of minutes after the return of an effective heartbeat, the pump-oxygenator may be an effective protection against dangerous air embolization. Bubbles released

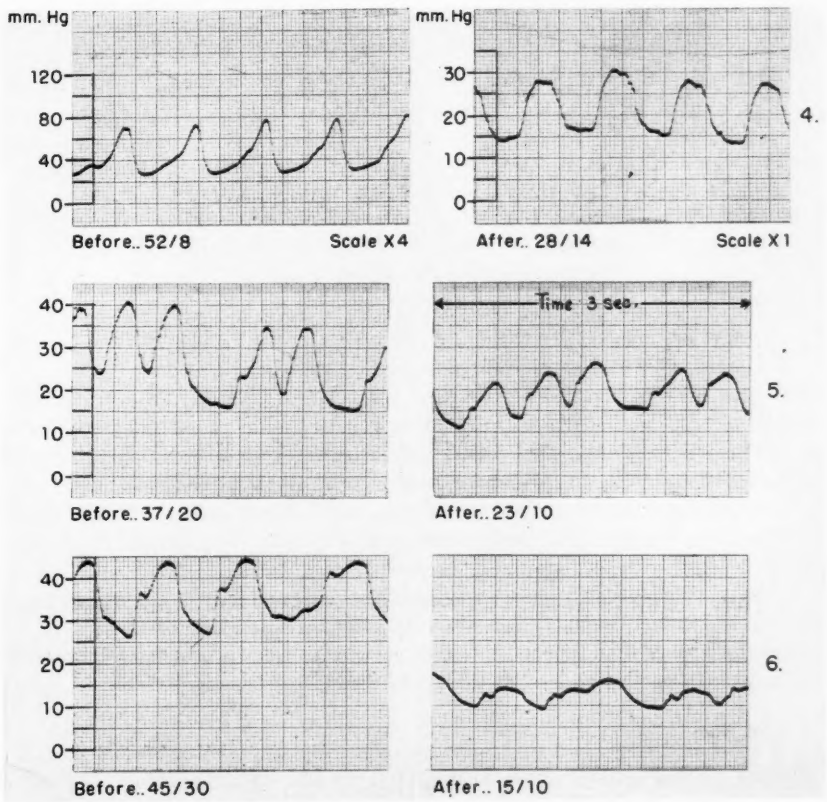

Fig 7.—Cont'd. CASE 4.—The left atrial pressures were considerably improved although not reduced to normal. (Note that record before correction is recorded on scale ×4.) Disappointing was the high pulmonary artery pressure before (68/44) and after the operation (64/28) indicating advanced pulmonary artery disease. (Patient died 3 weeks later.)

CASE 5.—Also some degree of mitral stenosis. The left atrial pressures, as recorded, were reduced. The mean left atrial pressures as recorded by the left atrial cannula which were reduced from 12 mm. Hg to 3 mm. Hg. (The fact that the mean pressures read lower was caused by a slightly higher base line. Also, by waiting 10 or 20 minutes, the left atrial pressure usually comes down further when the left ventricle has had a chance to recover.)

CASE 6.—Also considerable mitral stenosis. Very gratifying reduction in left atrial pressure after operation. At the same time the pulmonary artery pressure came down from 60/36 to 10/1; however, one should be careful in one's optimism in these results as the corresponding peripheral arterial pressure during the measuring before operation varied between 100 and 130 and afterward was only 80 mm. Hg.

from the beating left ventricle can produce disastrous sequelae if they reach the central nervous system. However, air bubbles released during high flow perfusion will be diverted into the coronary circulation. Although ventricular arrhythmia may follow, this is a transient complication and should not affect the ultimate outcome of the operation.

When perfusion has been discontinued, and the patient is maintained by his own heartbeat, pressures again are recorded for each chamber of the heart. If surgical benefit has been accomplished, there should be a significant drop in the left atrial pressure. In cases of severe mitral insufficiency, the pressures of the right ventricle and pulmonary arteries may be unaltered, even though the left atrial pressure has fallen a total of from 15 to 30 mm. Hg. In all probability this is explainable on the basis of fixed arteriolar resistance in the pulmonary vascular bed. In the majority of cases, this pulmonary resistance should gradually subside in response to lasting surgical improvement.

The immediately postoperative care is little different from that of any patient who has had open cardiotomy with total body perfusion. Since we have employed a single thoracotomy, complications usually are restricted to the right side of the chest. The patient should receive the care usually given to any patient who has severe disease of the mitral valve. In addition, of course, the surgical team must attend to the multitudinous details demanded by any operation utilizing perfusion of the entire body.

Results.—Time has not permitted proper evaluation of all patients included in this series. There have been 4 postoperative deaths. The first patient died after surgical fenestration of the mitral valve. The second, a 28-year-old man, who had iatrogenic insufficiency after surgical correction of mitral stenosis, died 6 hours after operation. The cause of death is attributed to diffuse interstitial pulmonary hemorrhages; left heart decompression had not been used. The other 2 patients, both adults with Class IV rheumatic heart disease, died 4 and 6 weeks after operation; congestive failure secondary to irreversible myocardial disease was the cause of death in both patients.

For the surviving patients, benefit has been assessed only by clinical means—none have been subjected to left heart catheterization. Four of these were operated upon more than 1 year ago—each patient has shown benefit by increase in tolerance of exercise and in reduction of the size of the heart.

DISCUSSION

The surgical attack upon mitral regurgitation, as we visualize it now, is palliative in nature. The limited experience and the short follow-up period do not allow proper evaluation of our efforts to date. It is quite unlikely that the measures now employed will prove to be curative in the strict sense. Treatment of an incompetent valve by distortion of the annulus or buttressing of a leaflet can hardly be called ideal. However, the incompetent mitral valve that has suffered the ravages of severe rheumatic fever is a poor subject for an ideal plastic repair. The myocardial and pulmonary damage together with the ugly valve of mitral insufficiency render the prospects of true cure dim indeed.

Nevertheless, an operation that offers quantitative reduction in the degree of insufficiency is of corresponding value. If the surgeon can reduce the systolic pressure of the left atrium by 50 per cent, it is likely that the patient will benefit appreciably. Whether the measures described herein can accomplish this over a long-term survival remains to be seen. Furthermore, we have misgivings about the fate of suture material of any type that must be subjected to the fatiguing combination of constant motion and the tension caused by left ventricular pressure. However, one thing is certain, the mitral valve is accessible by open cardiotomy utilizing the pump-oxygenator; and the patient with severe mitral insufficiency may tolerate such an operation if immediate benefit ensues.

There is another factor that detracts from the present operation for mitral insufficiency. While the surgeon is working directly on the mitral valve, either altering the annulus or reinforcing a leaflet, the extent of repair becomes a matter of judgment and experience. There is no measuring stick that can be used as a therapeutic guide while the heart is open and in a functionless state. Actually, the surgeon must wait until the cardiotomy incisions are closed, perfusion has been stopped, and the cardiac function has been restored, before he can estimate the degree of success. Even then, success is only partially gauged by the alteration in interatrial pressures after satisfactory rhythm is established. This delay in evaluation is in sharp contradistinction to the closed operation for mitral stenosis, in which the surgeon's index finger accurately assesses alterations in the valve during and after commissurotomy. The digital evaluation is performed within the functioning heart, and guides the surgeon in his decision to pursue the valvulotomy or to terminate the procedure. This type of "evaluate-as-you-go" procedure is not possible in open-heart operations for mitral insufficiency.

It would seem that the ideal operation for mitral insufficiency will consist of prosthetic-valve replacement. We are encouraged to believe that this will be feasible with the development of better prosthetic devices. Since the exposure already is adequate and the surgeon has ample time, with or without cardiac arrest, the next advance must be a technical one. In other words, the postulated cure of mitral insufficiency will involve a satisfactory valvular replacement that stimulates function of a normal valve.

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(For Discussion, see page 690.)

SURGICAL TREATMENT OF MITRAL INSUFFICIENCY

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IT HAS BEEN apparent during the developmental phase of mitral valve surgery that further progress would come only when the mitral valve could be visualized adequately and the various pathologic factors appraised, evaluated, and corrected under direct vision. This phase necessitated the successful adaptation of extracorporeal circulation to left heart lesions. Experience gained from the use of perfusion techniques in the correction of congenital right-sided lesions was beneficial. Additional factors, however, were present: such as the proper approach and exposure, the advisability of elective cardiac arrest, the prevention of air embolism, and the proper surgical techniques. Observations will be presented from the research laboratory, as well as experience gained from the direct vision correction of mitral valvular disease in 28 patients.

Pathology.—Mitral regurgitation is usually secondary to rheumatic involvement of the mitral valve, but may be congenital in origin. There are two main types of acquired mitral regurgitation—the pure and the mixed. In the *pure form*, the valvular pathologic changes are at a minimum. There may be some thickening of the cusps, some rolling of their edges, and some contraction of the cusps leading to a mild degree of regurgitation. Once the regurgitation has begun, a self perpetuating set of circumstances tends to worsen the process. Ventricular hypertrophy and later dilatation results from the heart's attempt to compensate for the lessened cardiac output from regurgitation. This in turn leads eventually to further dilatation of the mitral annulus and further regurgitation. The directional flow of blood toward the aortic outflow tract causes a greater degree of widening of the posteromedial commissure and separation of the cusps. The factor of myocardial insufficiency assumes greater importance as the condition worsens.

The *mixed type* is composed of varying degrees of stenosis as well as regurgitation. The other lesions of rheumatic fever are also evident. There is absolute loss of valve substance as a result of the cicatricial contraction of the valve leaflets. There are varying degrees of commissure obliteration giving rise to stenosis. The diseased chordae tendineae may be fibrotic and foreshortened

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which, in conjunction with the inversion of the leaflets into the ventricular chamber and adherence to the endocardium, causes the valve to have varying degrees of fixation in the open position. Again, in an attempt to compensate for the regurgitant flow of blood, the ventricle undergoes hypertrophy and dilatation, with consequent annular dilatation and aggravation of the pathologic process. All gradations between stenosis and regurgitation may be encountered from being predominant stenosis to predominantly regurgitant. As such, the valve orifice configuration, as noted in Fig. 1, may be teardrop, fishmouth, patulus, or wholly irregular from extensive loss of valve substance. We have been impressed with the greater degree of regurgitation noted visually over that

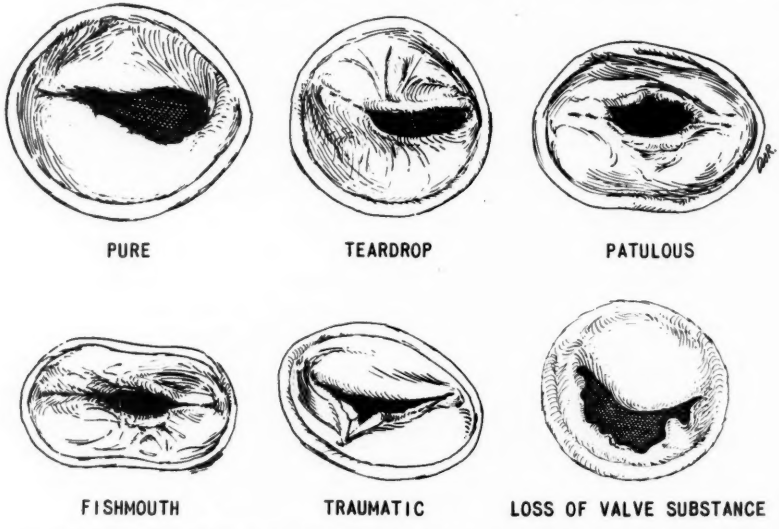

Fig. 1.—Diagrammatic sketch of variations in pathologic process in patients with mitral regurgitation.

detected by the intracardiac finger. Although the regurgitation takes place most commonly and to the greatest degree at the posteromedial aspect of the valve, it may be general or occur at the anterolateral aspect.

Traumatic regurgitation may result from an improperly executed commissurotomy. This was present in 1 patient in this series, in which the patient had undergone a commissurotomy at another hospital 2 years previously and had been worsened by the procedure as a result of an inadvertent incision in the lateral leaflet.

SELECTION OF PATIENTS

In the recent past, prior to an effective technique for the surgical correction of mitral regurgitation, considerable effort was exerted in the selection of patients for valvular surgery, directed at excluding patients with mitral regurgitation and multivalvular disease. Many diagnostic tests were employed to aid in this differentiation, including left-sided cardiac catheterization, Evans blue dye

dilution curves, and Diodrast studies. Although these examinations in skilled hands are not associated with any great degree of complication, they are still formidable and frequently exhausting to the sick cardiac patient, and not infrequently do not provide the desired information. Since we now have effective surgical techniques to cope with regurgitant lesions, as well as multivalvular disease, the need for this more precise preoperative information is less important in view of the fact that it can be readily obtained at the time of surgical exploration by pressure studies and direct examination.

The history, clinical course, and physical examination still remain a very important aspect of the appraisal. The electrocardiogram and roentgen examination give valuable information as to chamber hypertrophy and stress. It is particularly valuable to have previous studies available for comparison to determine whether significant change or progression of disease has occurred. Phonoelectrocardiograms are employed for comparison with postoperative studies. A definite improvement in the valve sounds is now being heard, which was a very infrequent occurrence in patients operated upon by closed techniques. Right-sided cardiac catheterization, with and without exercise, a simple and entirely safe technique, is routinely employed to determine the status of and changes in the pulmonary vasculature, as well as providing information regarding cardiac output and reserve.

Pulmonary wedge pressure studies obtained at this time not only provide an index as to left atrial pressures but also provide suggestive evidence as to the presence of regurgitation and/or stenosis, or both. It is important to determine the presence or absence of rheumatic activity by sedimentation rates and the presence of C-reactive proteins. A period of observation in the hospital of the temperature and pulse is important. Liver and kidney function tests are employed when indicated. In some instances, a longer period of observation is necessary to determine the patient's response to definitive therapy in the presence of failure. The likelihood of a successful result is not great in a patient showing progressive evidence of cardiac failure while on a strict medical regimen.

If the above studies show the patient to be symptomatic, to have an enlarged heart, to have electrocardiographic evidence of left ventricular and atrial hypertrophy, and evidence of pulmonary hypertension which is aggravated by exercise and does not have signs of activity, then operation is recommended; particularly, if serial examinations reveal a definite progression in such findings over previous examinations. The cardiopulmonary reserve is great in patients with mitral regurgitation—once there is evidence of progression of the above findings, we believe it is unwise to postpone operation to await more urgent indications because of the detrimental effect on the myocardium which may compromise an otherwise successful result.

OPERATIVE APPROACH AND EXPOSURE

The mitral valve can be adequately exposed from either the left or right side. A left-sided exposure was used in four instances and a right-sided exposure in twenty-four. The 4 patients in whom a left-sided exposure was used

had only a small area of left auricle presenting on the right side. It was felt initially that this might limit the length of the incision in the auricle and in turn impair visibility if the mitral valve were to be exposed from the right side in such instances. Subsequently, it has been found that an adequate exposure can be obtained from the right side in spite of the amount of left auricular wall presenting into the right hemithorax.

A right anterolateral incision with splitting the sternum and with the right shoulder elevated 20 to 30 degrees, was used in twenty-one instances and a posterolateral approach in three. We prefer the right-sided anterolateral approach for to us it appears easier and faster. It requires a smaller incision and is associated with less blood loss and operative trauma. A more adequate exposure of the aortic valve is obtained if operative correction of this valve is also required than is possible in a posterolateral approach. Furthermore, in the advent of cardiac arrest or ventricular fibrillation, access to the heart is more readily obtained. Exposure and cannulation of the femoral vessels is easier from this approach than in a posterolateral one in that with us this aspect of the operation proceeds concomitantly with the thoracotomy. If an extreme posterolateral incision is employed, the aortic valve becomes rotated to the left anterolateral aspect of the mitral valve so that there is less room in the ventricular outflow chamber for the collection of air. However, this is felt to be an unimportant aspect in the etiology of air embolism.

ADVISABILITY OF CARDIAC ARREST

Feeling initially that there was greater safety from air embolism with the use of cardiac arrest, this was utilized in the 4 patients operated upon from the left side and in the first 3 patients operated with the right-sided approach. In one instance, in which a left-sided approach was employed, evidence of transitory cerebral air embolism was noted postoperatively. We do not feel that the use of elective cardiac arrest is a factor in the prevention or etiology of air embolism if the other precautionary measures are employed.

To correct mitral insufficiency surgically the surgeon must have a knowledge not only of the structural changes present but also of the relationship between structural changes and functional changes. Functional changes in the case of valvular disease means motion—the proper opening and closing of the valve. Nothing short of observing the valve mechanism in the beating heart will allow the surgeon information as to the pathologic physiology of that patient's valvular mechanism. With the heart beating, the factors contributing to the regurgitation can be properly assessed and evaluated; the location of the regurgitation—by no means constant—can be determined and the effectiveness of the correction noted. In several instances, annular plication at one site distorted calcified valves so that regurgitation occurred elsewhere. In such instances, visualization of this regurgitation in the beating heart allowed further repair and obviated a partial or poor result.

Other advantages of allowing the heart to beat are that such operations need not be hurried. The impaired myocardium is kept well oxygenated and

there is no danger of the occasional difficulty in restarting the heart which may be encountered in patients in whom the myocardial reserve is poor and which may be harmed further by the detrimental effects of potassium or anoxia. There has been no apparent harm resulting from the use of temporary or intermittent aortic occlusion if employed during the procedure to allow a drier field during the initial placement of the sutures in the event of considerable aortic and ventricular regurgitation.

AIR EMBOLISM

Laboratory experience showed us that the mitral valve could be approached from the right side, with the heart beating, without a high incidence of air embolism. Also, there had been no evidence of air embolism in our clinical experience in patients with atrial septal defects, atrioventricularis communis, and transposition of the great vessels, corrected by atrial septal transplantation. These patients were also positioned in a similar manner with the right side of the body elevated approximately 20 to 30 degrees with the opening in the auricle higher than the mitral valve.

In spite of the above, 1 patient approached from the left side with cardiac arrest, and 3 patients approached from the right side allowing the heart to beat, had evidence of air embolism postoperatively. Two of these patients had a right anterolateral incision and the third a right posterolateral incision. Three of the 4 patients had primarily stenotic rather than regurgitant valves. The 1 approached from the left side was primarily regurgitant. In 1 patient the escape of air into the aorta was apparent even before the blood in the auricle was entirely aspirated and the valve visualized. Two successive ventricular diastoles sucked the auricle not only dry of its blood content but also sucked in air. In such instances, the resistance of the stenosed mitral valve may be greater than the systemic pressure against the aortic valve which, in the presence of a forceful ventricular contraction, may allow the ventricle to pump air and foam into the aorta. Evidence of air embolism was not encountered in patients having predominant mitral regurgitation, except for the 1 noted above. Because of these incidents of air embolism, the problem was again studied in the laboratory.

Previously in our laboratory work, evidence of air embolism was noted only at the conclusion of the operation and the particular phase of the operation, or technical maneuver, that perhaps caused it, could not, therefore, be assessed properly. A series of experiments were conducted in which a glass tube with bubble trap was inserted into the arch of the aorta or into the brachiocephalic artery so that the presence of air could be immediately detected (Fig. 2). The various maneuvers incident to mitral valve correction and total body perfusion were then duplicated with the following observations.

1. The mitral valve could be exposed; the left auricle and left ventricle could be emptied of its blood content and no air would enter the systemic circulation as long as the mitral valve was held open and ventricular systolic pressures were prevented from exceeding aortic diastolic perfusion pressures (Fig. 3). If the aortic valve, however, was partially opened, either by the finger

or by undue tension upon the aortic cusp of the mitral valve, then air occasionally escaped into the aorta, even in the presence of aortic regurgitation. This was not a constant finding.

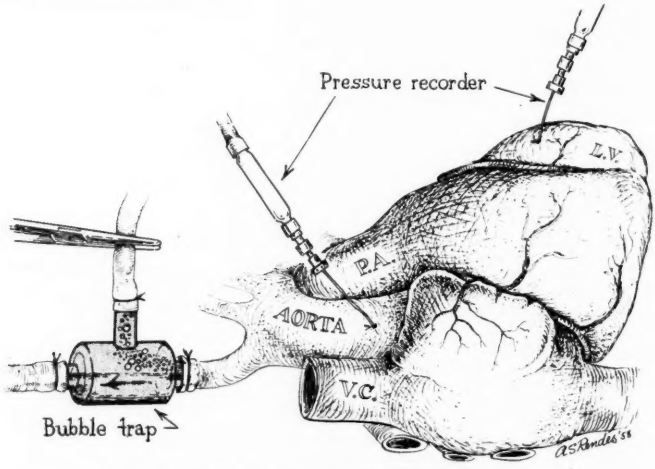

Fig. 2.—Various factors contributing to air embolism detected experimentally by means of bubble trap in brachiocephalic artery and pressure manometers in aorta and left ventricle.

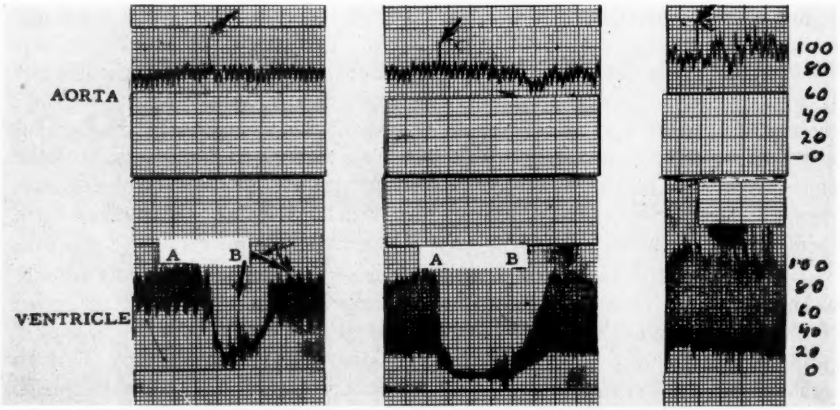

Fig. 3.—Factors Contributing to Air Embolism. Manometer tracing demonstrating absence of air embolism as long as mitral valve is kept partially open so that ventricular systolic pressure never exceeds aortic diastolic perfusion pressure. *A*, Valve closed. *B*, Valve open.

2. A small collection of air could collect in the ventricular outflow chamber adjacent to the aortic cusps which might not be regurgitated into the auricle upon filling. If the ventricle and auricle were allowed to fill with blood for closure without aspirating this air, then, as soon as the mitral valve was allowed to close, the ventricular beat would become effective and this air could escape into the systemic circulation.

3. If the mitral valve was kept open, allowing blood to collect in the ventricular chamber and auricle, foam in the ventricle would be washed back into the auricle. As soon as the air bubble in the ventricle, when present, was aspirated, and the foam had escaped or had been aspirated, the instrument holding the mitral valve open could be removed when the auricle was filled with blood and the auricular incision closed without evidence of air embolism.

4. If the aorta were clamped because of regurgitation, the aortic valve may open and allow air to collect proximal to the clamp. Consequently, such a clamp should be placed close to the aortic valve rather than nearer to the arch to eliminate this potential air space. In case of doubt, one should aspirate the base of the aorta for air while releasing the clamp.

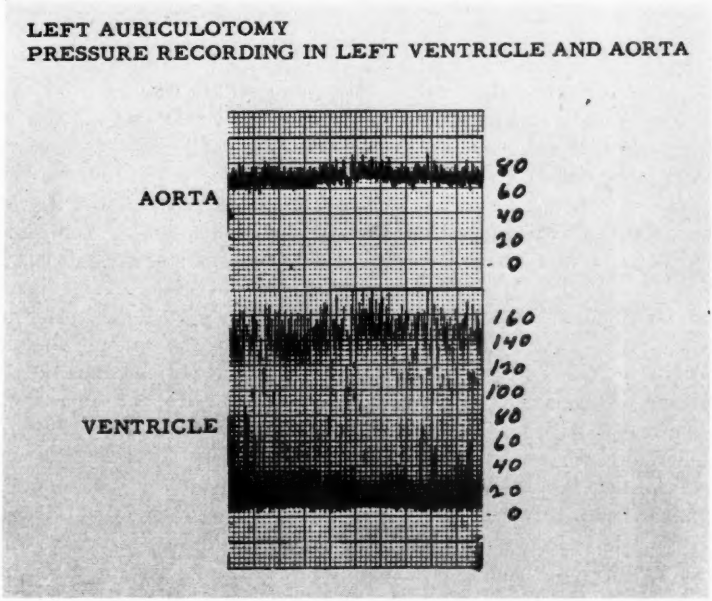

Fig. 4.—Factors Contributing to Air Embolism. Manometer tracing demonstrating presence of air embolism when circumstances allow ventricular systolic pressure to exceed aortic perfusion pressure.

5. By simulating mitral stenosis by partially occluding the mitral orifice by suture, keeping the small residual stoma open, or with the mitral valve closed allowing blood to be regurgitated into the ventricular chamber, and with a pressure recorder in the ventricular chamber and aorta, air could escape during the diastolic aortic phase, if the ventricular systolic pressure exceeded the diastolic aortic pressure resulting from a low aortic perfusion pressure or a highly pulsatile type of arterial pump (Fig. 4). Consequently, a high perfusion pressure should be maintained and a nonpulsatile pump, or a pulsatile pump with only minimal phasic differential in pressure, should be employed to ensure a constant pressure against the aortic valve.

During the surgical correction of mitral insufficiency, the effectiveness of closure of the mitral valve is so inefficient during the early phases of the technique that ventricular systolic pressure can never become great and is easily dissipated in a retrograde direction. As the effectiveness of the correction proceeds, and as the regurgitation lessens, the resistance in the mitral valve may become sufficiently great so as to allow the ventricular systole to push air and foam into the aorta. Consequently, as the mitral valve becomes more competent or as the effectiveness of repair is being evaluated, an area in the valve region that is competent should be kept partially open to prevent the left ventricle from obtaining systolic pressures effective enough to pump air or foam into the systemic circulation. This instrument should keep the mitral valve partially open until the completion of the repair, until all the foam and air have been aspirated from the ventricle, and until the blood has largely filled the auricular chamber.

Three of the 28 patients undergoing direct vision correction of mitral deformities, who had transitory evidence postoperatively of cerebral air embolism, had varying degrees of mitral stenosis. Although the valve was mainly stenosed, a rigid opening or stoma remained through which air and blood could be sucked during ventricular diastole. The resistance of this stenosed valve during ventricular systole was greater than the pressure against the aortic valve, so air and foam were apparently discharged into the systemic circulation. In contradistinction to patients having primarily regurgitation, those with stenosis are more likely to have evidence of air during the initial phase of the operation and this danger continues throughout the period that the mitral valve is exposed prior to the commissurotomy. To minimize this hazard, the mitral valve should first be explored digitally. If significant stenosis is present, it should be partially relieved digitally and an instrument inserted to maintain the valve in an open position before the auricle is emptied of its blood content for completion of the commissurotomy and correction of the regurgitation.

If the auricle is filled with clot, as it was in the three instances, or if the mitral valve is so highly calcified that the danger from calcium embolism from the digital opening is too great, then the aorta should be clamped at its base before these mitral maneuvers are undertaken. As soon as the clots are removed, the commissurotomy performed, and the loose calcium removed from the valve, an instrument can then be inserted to hold the valve leaflets apart; the anesthetist temporarily occludes the common carotids, and the aortic clamp is slowly removed. It is important in such instances that a filter be employed in the sump circuit to avoid embolization through the machine.

TECHNIQUE

A rotating disc oxygenator with a Sigmamotor pump was used initially. Recently a roller pump has been used (Fig. 5). Occasionally, but not routinely, the right auricle is cannulated for coronary sinus blood to return to the machine. Because of the extraordinary amount of blood that may be regurgitated into the auricle even in the presence of a normal aortic valve, it is necessary to have adequate sump facilities to handle as much as 2 to 3 L. of blood if necessary. We have employed two sumps—one with a filter system—to be used

in the event of considerable calcium deposits or clot, and the other without. Two bubble traps are available to handle this increased sump load, if necessary.

A flow rate of 50 c.c. per kilogram per minute which results in an arterial inflow of 3 to 4 L. per minute has sufficed to maintain a mean arterial pressure of 70 to 80 mm. Hg. The inflow cannula is always in the common femoral artery through the superficial femoral artery. The venous return to the oxygenator has been by pump suction regulating the venous return by maintaining the

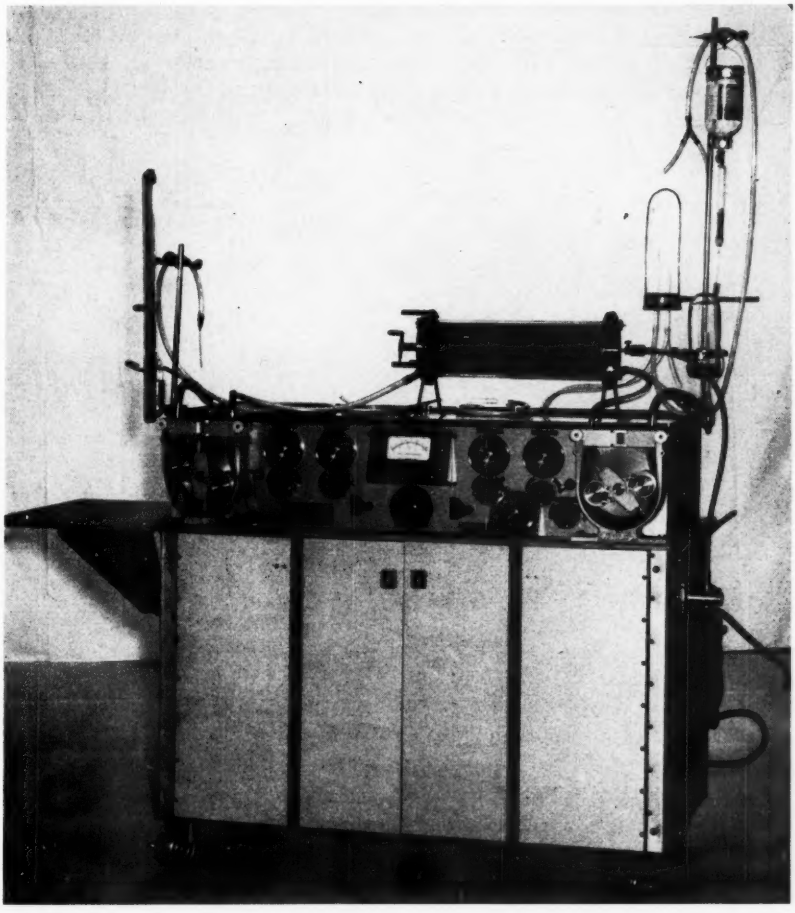

Fig. 5.—Picture of the Kay-Cross rotating disc oxygenator used in conjunction with a roller pump mechanism.

vena caval pressure at pre-perfusion levels. This, we feel, gives us a better control over body blood volume. Constant monitoring of the mean arterial pressure, mean vena caval pressure, electrocardiogram, electroencephalogram, pO_2, pCO_2, and pH, body and oxygenator temperature provide us with a constant

physiologic status of the patient. Hematocrit, hemoglobin, and body weight are determined immediately prior to and following surgery. The patient is placed on a heated mattress with the right side elevated between 20 to 30 degrees.

As an anterolateral incision with transection of the sternum is being made, the femoral cannulations for arterial input, arterial manometer, and intravenous infusion are being made simultaneously.

In case of question, the status of the tricuspid valve is determined by digital exploration. Pressure recording of the aorta and left ventricle are then obtained to verify or eliminate coexistent aortic valvular disease. Pressure recordings of the left auricle are then made. The extracorporeal perfusion is then begun while the left auricle is incised and the mitral valve digitally explored. If the mitral valve is obviously incompetent, the sump then empties the left auricle of its blood content so the various factors contributing to the regurgitation can be

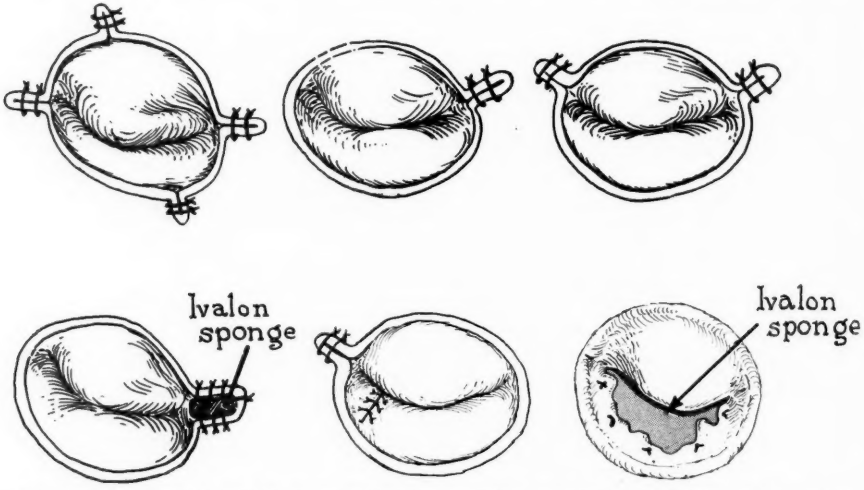

Fig. 6.—Diagrammatic sketch of variations in technique of annular plication and valvular substitutions employed in the surgical correction of mitral insufficiency.

further visualized and assessed. If a significant degree of stenosis is also present, the valve is opened as widely as possible digitally and an instrument is inserted between the valve leaflets prior to aspirating the blood from the auricle. The commissurotomy is then completed under direct vision. This is followed by visualization of the valve cusps and chordae tendineae. In patients with pure mitral insufficiency, it has been necessary not only to plicate the annulus at both the commissures but also anteriorly and posteriorly (Fig. 6). As the valve becomes more competent, it is important to keep an area of the valve separated to allow regurgitation as other aspects of the valve are being evaluated. Several mattress sutures should be used in the plication of each area so that an adequate apposition of the tissue is forthcoming. The sutures should be without tension.

In patients with mixed mitral stenosis and regurgitation, it is important that a complete commissurotomy and valve leaflet mobilization be accomplished prior to correction of the insufficiency for otherwise an extensively calcified

commissure prevents, or may prevent, annular approximation of the valve by plication. If a highly calcified commissure or cusp prevents cuspal approximation, the residual opening can be obliterated by incorporating a plug of Ivalon in the plication. Occasionally, it is possible and beneficial to remove the bulk of this calcium prior to the annular plication.

In an occasional patient, one of the mitral cusps—usually the mural—will be so destroyed from the rheumatic involvement as to lack adequate valvular substance. In such instances, valve substitution in the form of a sheet of compressed Ivalon (10 to 2) can be incorporated on the ventricular aspect of the cusp as demonstrated in Fig. 6.

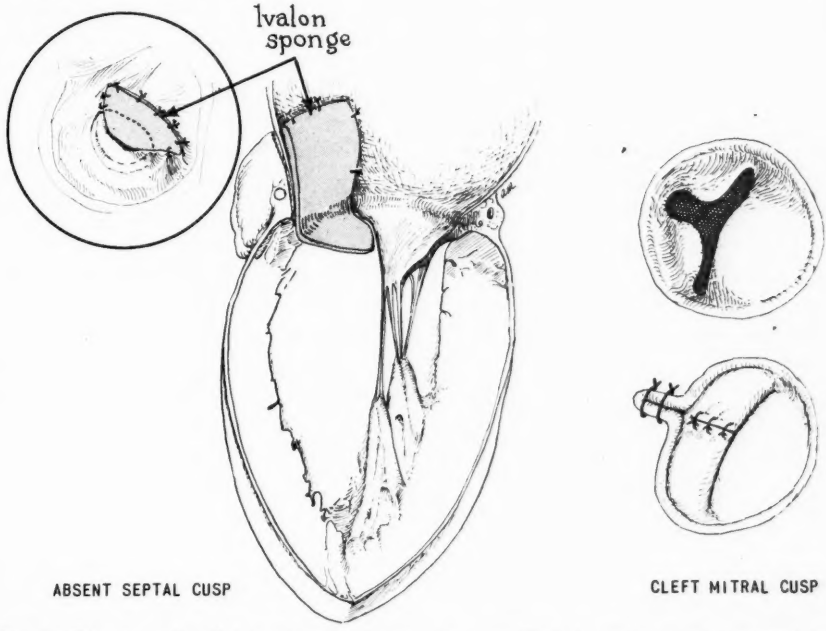

Fig. 7.—Diagrammatic sketch of surgical correction employed in patients with congenital mitral regurgitation associated with atrioventricularis communis.

In 1 patient the regurgitation present resulted from an inadvertent cut into the posterior leaflet below the lateral commissure by a commissurotomy knife during a previous operation at another hospital. Four interrupted mattress silk sutures were employed to sew the torn cusp. A commissurotomy was then performed, followed by an annular plication at the posteromedial commissure. This patient had an excellent result and is completely free from any murmur.

CONGENTIAL MITRAL REGURGITATION

Congenital mitral regurgitation is usually associated with atrioventricularis communis. Two forms appear to exist. The more common form is a cleft anterior cusp which is in reality probably the division between the common anterior

and posterior atrioventricular cusps of the mitral and tricuspid valves. The other form noted has been the absence of an anterior cusp or the presence of a very rudimentary cusp. The septal aspect of the annular system of both valves may be defective allowing the further dilatation of such valves.

Success of surgical correction is not only dependent upon the closure of the septal defects without tension but the effectiveness with which the valvular incompetence can be corrected. In the event of a bifid or cleft cusp, both the mitral and tricuspid aspects should be approximated by several interrupted sutures. Valvular approximation should also be aided by approximating the anterior and posterior aspects of the annular system at the septal level.

When the septal cusp is so rudimentary as to be entirely ineffective, the closing mechanism of the mitral valve results from closure of the mural leaflet against the ventricular septum. With closure of the septal defect, a shift of the septum to the right results, causing further incompetence of the mitral valve. Competence of this valve in one instance appeared to be effected by fashioning an Ivalon septal cusp in continuity with the Ivalon patch used in closure of the septal defect, as illustrated in Fig. 7.

RESULTS

The incompetent valves in the majority of patients with mitral regurgitation can be surgically corrected by the techniques described. The ease and effectiveness of the correction is largely dependent upon the severity of the pathologic process. Valves composed of severely matted cusps and chordae tendineae, loss of valve substance, or those which are highly calcified, are the most difficult to repair and, short of valve replacement, the surgeon in some instances must be content with a result short of ideal, obtaining as much improvement as possible by the plastic procedure and the addition of the plastic prostheses that are available at the present time. The role of chronic myocarditis, myocardial failure, and the presence of other valvular defects also are important factors in the eventual result. Fortunately, the pathologic process in the majority of the valves is such that they can be corrected and the myocardial reserve is usually sufficiently great as to provide satisfactory function. Fortunately, the number of patients whose process has extended beyond surgical redemption will be few. With continued progress in this field, earlier surgical intervention, and probable lessened severity of the pathologic process from improved therapeutics, greater benefit can be anticipated in the future.

It is too soon to attempt an evaluation of the surgical results obtained in patients with mitral regurgitation corrected by the open technique, for this group have only been operated upon within the past year. However, comparison with the early results obtained previously by closed methods allows for considerable enthusiasm for the open technique at present. The eventual results will be dependent not only upon the effectiveness and permanence of surgical repair, but also upon the myocardial reserve and the reversibility of pulmonary vascular changes. The disease process in the majority of patients was sufficiently

severe as to cause left ventricular hypertrophy, left atrial enlargement, severe pulmonary hypertension, and right ventricular hypertrophy. With reversal or improvement in the pulmonary vascular changes with further time, greater benefit might be anticipated at a later date.

The marked improvement in atrial pressures obtained immediately postoperatively (Table I), the rapidity of the convalescence, the decrease in heart size, and the rapid increase in exercise or work tolerance in the majority of patients following operation, all point to a gratifying eventual result. Routine cardiac evaluation studies, including cardiac catheterization, are obtained 6 months to 1 year following surgery. Time has not allowed the complete performance of these studies as yet.

TABLE I. MITRAL REGURGITATION

PATIENT	PRESSURE RECORDING LEFT AURICLE (mm. Hg)	
	PREOPERATIVE	POSTOPERATIVE
J. W.	30/25	18/4
V. B.	68/46	12/4
M. L.	42/19	22/15
F. B.	70/22	36/16
C. M.	60/24	16/7
J. P.	16/12	4/2
P. C.	36/20	16/8
S. T.	26/11	8/4
E. H.	26/22	10/4
J. S.	30/17	20/8

There were six deaths in this group, an operative mortality of 20 per cent. An analysis of the cause of death in these patients shows that in the future with greater experience some of these deaths can undoubtedly be avoided. Failure, in the first patient to be operated upon, was thought to be due to the presence of a severely calcified and destroyed valve, a poor perfusion, and a blood imbalance at the end of the operation. Two patients died immediately postoperatively from what was felt to be poor myocardial reserve with myocardial failure. One patient died from either air or calcium embolism. One patient died from an electrolyte imbalance and renal failure. This patient had a longer perfusion period than usual due to the inability to maintain a satisfactory cardiac output. An atrial septal defect was later found that had not been detected previously. As soon as this was closed the heart was then able to maintain a good output. The cause of death in the other patient was due to an electrolyte imbalance with retention of fluid, abdominal distention, and pulmonary edema. In the future, it is felt that complications due to air embolism can be prevented and those due to electrolyte imbalance largely overcome.

COMMENT

It is felt that with the use of a mechanical pump-oxygenator, direct vision correction of mitral regurgitation will now be a realization in the majority of such patients. The valves in a few patients are so destroyed that nothing short of valve replacement will suffice. The operative mortality and morbidity during

the developmental phase has been low. Some of the technical factors contributing to this mortality and morbidity are correctable. Others, such as pulmonary vascular changes and poor myocardial reserve, can also be largely avoided by earlier surgical intervention.

SUMMARY

Surgical correction of mitral regurgitation can now be satisfactorily accomplished in the majority of patients with mitral regurgitation. The selection of patients for surgery based on the cardiac evaluation studies is discussed. The aspects of the surgical technique, including the approach and exposure, the advisability of elective cardiac arrest, the prevention of air embolism, and the correction of the incompetent valve are described. Factors contributing to the operative mortality and morbidity, the majority of which can be corrected or prevented, are discussed. The initial results have proved to be exceedingly satisfactory, and, with further experience, should be even more gratifying.

Discussion

(Papers by Effler [page 665], Kay [page 677] and Their Associates)

DR. H. T. NICHOLS, Philadelphia, Pa.—I have enjoyed the papers of Dr. Effler and Dr. Kay very much. I rise merely to mention our experience with 92 operated cases of mitral insufficiency. The surgical technique has been essentially the same in all cases and was reported to this Association in 1956.

(slide) This illustrates the method of affixing one part of the mitral annulus firmly to the other.

(slide) This shows the sutures tied into position.

The operative mortality by the original closed technique rose subsequently to 27 per cent. We then switched to the open technique in some of the poor risk patients, but lost 4 out of 5 so operated upon.

The majority of the mortality in our patients operated upon by the closed technique seemed to occur during the intracardiac manipulation required in applying the plicating sutures. The cardiac output seemed to be reduced by the surgical maneuvers with resultant decrease in coronary flow leading eventually to cardiac arrest or ventricular fibrillation. More recently, we have employed partial cardiopulmonary bypass with a closed technique in an attempt to get through this most critical part of the operative procedure.

The left thoracotomy approach is used. The uptake catheter to the extracorporeal circulation is inserted into the outflow tract of the right ventricle and the output catheter is inserted into the right femoral artery. We have now operated on 10 patients by this technique with no mortality in the operating room and one late death about 3 weeks postoperatively.

DR. FREDERICK S. CROSS, Cleveland, Ohio.—I would like to compliment Dr. Kay and Dr. Effler on what amounts to pioneer work in this rapidly advancing field. Frequently, the clinical achievements advance so rapidly, however, that we fail to appreciate some of the basic principles involved in a particular surgical problem. I would certainly agree with Dr. Kay that the important considerations in evaluating techniques for open mitral valve surgery are the accessibility of the valve, the incidence of air embolus, and the necessity for adjunct cardiac arrest. We have worked on this problem in the surgical research laboratories at St. Luke's Hospital in Cleveland and our experimental findings have been substantiated by recent clinical experience.

(slide) On looking at the location of the mitral valve superimposed on the cardiac outline, it is noted that this valve is some distance within the heart when approached from

the right side. This may not be too important when dealing with mitral insufficiency associated with an extremely large left atrium which affords adequate exposure. However, in mitral stenosis with a fairly normal left atrium, proper exposure can be a problem. When the mitral valve is approached from the left side through a transsternal incision, the operator may be nearer his work but, as Dr. Effler stated, the valve is still difficult to visualize because of the dependent position of the left atrium. Visualization of the valve is made more difficult by the fact that the cardiac apex tends to elevate when the pericardium is opened widely. This difficulty can be obviated to a great extent by stopping the heart.

(slide) More important than proper exposure, when considering any approach to the mitral valve, is the risk of air embolus from residual trapped in the left ventricle. The slide shows how an air bubble might be trapped in the left ventricular outflow tract with the patient in the supine position. Without taking special precautions, this air bubble is difficult to remove during or after cardiotomy closure.

(slide) The next slide shows the relative position of the cardiac chambers in the various approaches to the mitral valve. With the patient supine, the heretofore usual position for open cardiac surgery, it is noted that much of the left ventricle is superior in position to the left atrium affording the possibility for air to be trapped in the left ventricle. This potential for the trapping of an air bubble in the left ventricle is present with all of the left-sided approaches. However, as noted from the slide, this is not true when the mitral valve is approached from the right side. All portions of the left ventricle are dependent in position and it is a simple matter to let the left ventricle and left atrium fill with blood prior to the closure of the myocardiotomy. By keeping the mitral valve open as the left atrium fills, no air can accumulate in the left ventricular outflow tract. It is forced out of the cardiotomy incision placed in the superior position. The trapping of air is thus avoided. This is true whether or not the heart is approached through the right lateral or the right anterolateral approach. In addition, the cannulations for the heart-lung bypass are easily made from the right side, the other valves are easily approached if necessary, and there is the added dividend that adjunct cardiac arrest is not necessary.

Experimentally, with the right-sided approaches to the mitral valve, there was no instance of air embolus. Air embolus was a common occurrence, however, with the left-sided approaches; but, it could be prevented by placing a vent connected to suction in the left ventricle during and immediately after closure of the myocardiotomy. It would seem then at the present time the right-sided approaches to surgery of the mitral valve are the most desirable.

DR. ARTHUR VINEBERG, Montreal, Quebec.—A 25-year-old theater usher was admitted to the Royal Victoria Hospital on March 8, 1958. He had had fever and joint pains at 9 years of age without other indications of rheumatic fever. A second similar episode occurred at 14 years of age. Following this, he was asymptomatic except for inability to take part in athletics. In 1952, he was forced to stop work as a truck driver because of easy fatigability and shortness of breath. From this time, exercise tolerance progressively diminished and he was able to work only occasionally.

In 1955, he suffered an embolus to the right popliteal artery which did not produce ischemic changes. Later the same year he had a cerebral vascular accident with right hemiparesis and aphasia. Recovery from this complication was slow but complete. He was digitalized on this admission and maintained on digitalis afterward.

Physical findings included a blood pressure of 130/75 mm. Hg and an irregular apical pulse with a pulse deficit at the wrist. There was a marked apical thrust visible on systole. The heart was grossly enlarged to the left. There was a loud systolic murmur at the apex with radiation to the axilla, accompanied by a thrill. There was an aortic diastolic murmur. There was no other positive physical findings.

X-rays (Fig. 3, *A*) showed marked left ventricular and left auricular enlargement. Bullous cysts were visible at both lung bases. The electrocardiogram (Fig. 1) showed left ventricular hypertrophy with some right ventricular hypertrophy and atrial fibrillation.

He was considered to have rheumatic heart disease with predominant mitral insufficiency. Right heart catheterization was done. There was no indication of an intracardiac shunt. Pressure measurements of interest included the following ones.

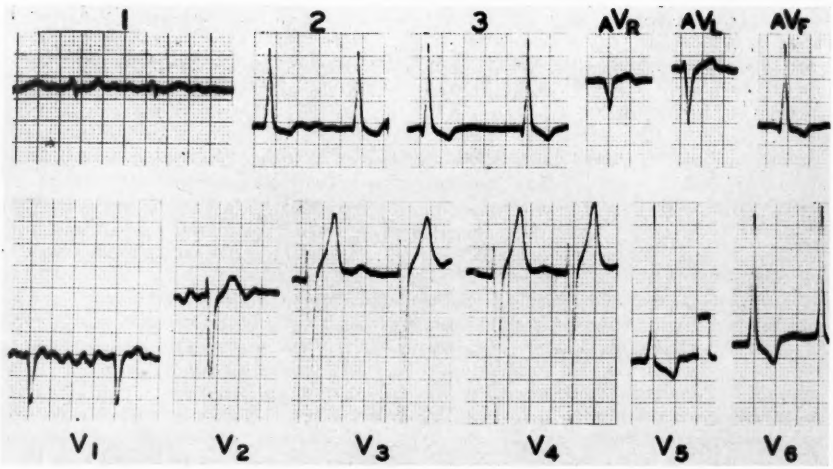

Fig. 1.—Electrocardiogram showing left ventricular hypertrophy with some right ventricular hypertrophy and atrial fibrillation. There was an aortic diastolic murmur.

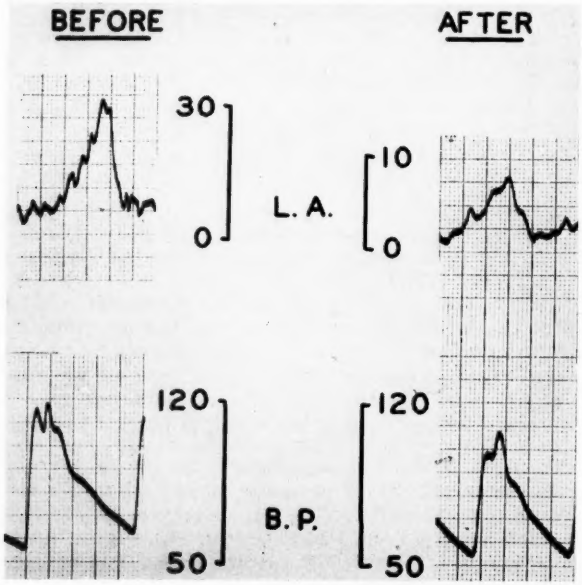

Fig. 2.—Left atrial pressures recorded in millimeters of mercury taken in the operating room before and after mitral annuloplasty which was performed to correct mitral insufficiency (L.B.). Note the marked drop in left atrial pressures after correction of the mitral insufficiency with systemic blood pressures approximately the same.

	Resting	*Light Exercise*
Pulmonary artery	37/18 (25)	65/40 (50)
Pulmonary capillary	20/12 (15)	60/20 (30)

On March 25, mitral annuloplasty was carried out through a right posterolateral thoracotomy. The pleural cavity was obliterated by adhesions. The pericardium was opened

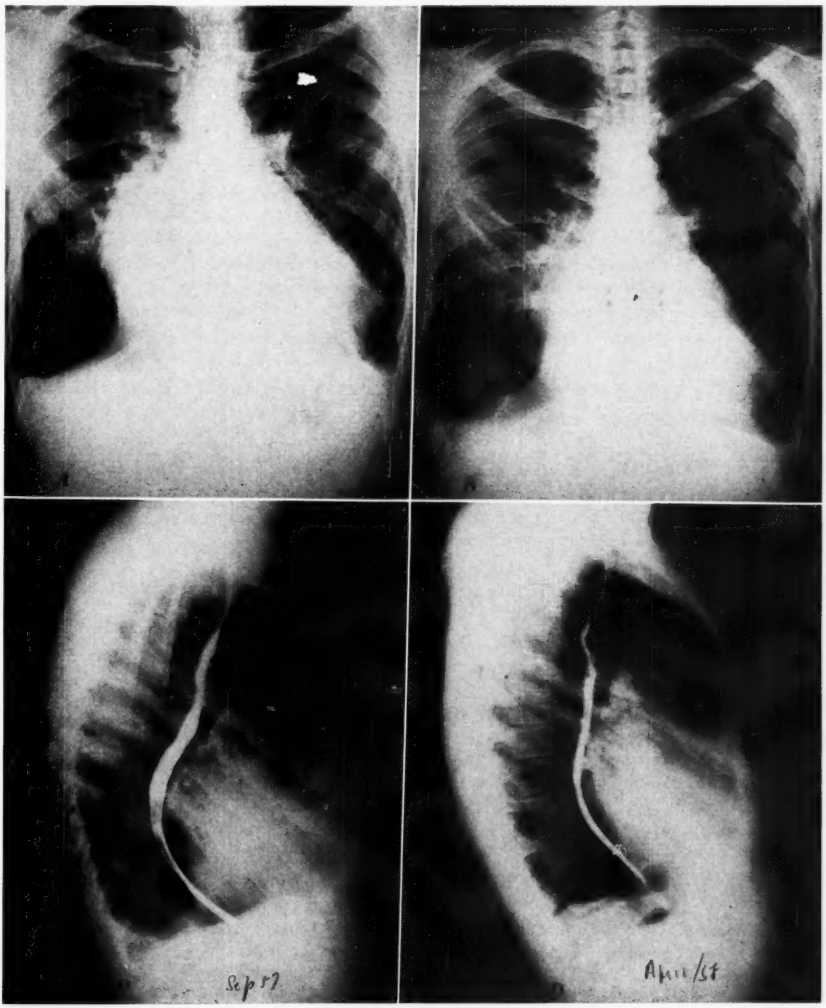

Fig. 3.—*A*, A P.A. roentgenogram prior to mitral annuloplasty. Note the markedly enlarged left atrium and left ventricle, also the presence of large lung cysts at both bases.

B, Shows a P.A. film of same patient 25 days postoperatively. Note the marked decrease in the transverse diameter of the heart which at this stage measured 2.6 cm. less than before operation.

C, A lateral view prior to operation showing left atrium pushing the esophagus posteriorly.

D, Lateral roentgenogram 22 days postoperatively showing marked decrease in size of left atrium which has greatly reduced the indentation of the esophagus by the left atrium.

and catheters were inserted into the superior and inferior venae cavae for return of blood to the screen oxygenator. The right femoral artery was cannulated for the return of arterial blood from the pump-oxygenator. Following the institution of extracorporeal circulation, the left atrium, which bulged well over to the right, was incised. The mitral valve was visualized and palpated. The outstanding finding was the great dilation of the annulus into which the palm of a hand could have been placed. Twelve No. 2 silk sutures were placed across the posteromedial aspect of the annulus in order to narrow the annulus to the point where the cusps of the valve could meet. The regurgitant jet, which was marked prior to placing these sutures, was eliminated by them. The left atrial incision was closed with silk sutures.

The bypass was continued for 45 minutes with a mean flow of 3,500 c.c. per minute. Left atrial pressures were taken before and after bypass (Fig. 2) and showed a drop to the normal range with systemic arterial blood pressure at comparable levels.

His postoperative course was marked by a swinging fever in the first week. This returned to normal thereafter without evidence of infection. Blood cultures were negative; otherwise, convalescence has been uncomplicated. There is definite reduction in the apical thrust and the patient himself is much less aware of the pounding of his heart. A systolic murmur remains audible at the apex. The electrocardiogram is unchanged. The x-ray shows 2.6 cm. reduction in heart size. (Fig. 3, *A, B, C, D.*)

The case reported is one of mitral insufficiency treated by open-heart surgery. Reduction in left atrial pressure has been demonstrated. The patient's course since operation has been gratifying.

DR. C. WALTON LILLEHEI, Minneapolis, Minn.—The advent of cardiac bypass techniques has completely changed the outlook for patients with advancing disability due to mitral insufficiency.

In the congenital variety of mitral insufficiency associated with the common atrioventricular canal, recongnition and correction are not difficult. In that regard, I should like to emphasize briefly two important points at some variance from what has been said today. This is based upon experiences with 41 patients operated upon by perfusion techniques since 1954.

First, cardiac arrest is entirely unnecessary in the direct vision repair of these defects, and, in fact, is actually detrimental.

The surgical exposure is excellent through a right atriotomy and, with the ventricles beating, it is far easier to locate accurately the pathology. The clefts in the mitral or tricuspid valves are immediately evident to the eye and their nature can be verified merely by allowing some of the coronary sinus blood to accumulate in the beating ventricles or, if necessary, some 5 per cent glucose can be poured into the ventricle. Other dividends of not arresting the heart are the avoidance of myocardial hypoxia or necrosis and the ability to note and immediately remove stitches obviously interfering with cardiac conduction.

Our second point relates to the method of repairing the clefts in the mitral or tricuspid leaflets. Simple interrupted stitches of fine silk are placed close together between the adjacent halves of the abnormally separated leaflets and are not tied but tagged temporarily with a mosquito forceps until the entire row necessary for the repair has been completed. Then, before tying, a slender strip of Ivalon (2 to 3 mm. wide and 3 to 4 inches long) is trimmed from a block of compressed polyvinyl sponge (5 to 1) and placed between the row of interruped stitches so that the knots can then be tied and seated firmly, but without tension, upon this thin strip of polyvinyl sponge. This results in a snug, blood tight closure upon which the left ventricular thrust is evenly distributed without tissue tension and, in addition, the Ivalon provides a source of reaction that ensures union between the approximated leaflets.

Again, the fact that the ventricles have not been arrested is helpful in evaluating the adequacy of the repair before turning attention to correction of the ostium primum septal defect.

Acquired mitral insufficiency, on the other hand, results from varying combinations of two primary factors: (1) an absolute loss of valve substance from cicatricial retraction and shrinkage of leaflet tissue and/or chordae tendineae, and (2) dilatation of the annulus fibrosus.

One of the very favorable aspects of pure mitral insufficiency lesions has been the consistent finding of relatively good leaflet tissue even in the far-advanced lesions. Calcium, although it may be found often in the left atrial wall opposite the regurgitant jets, is rarely present in the leaflets. Moreover, the latter, although somewhat thickened and incompetent, remains supple and flexible. These findings are in marked contrast to the usual pathology of the purely stenotic lesions.

It is evident to us that the element of annular dilatation has been greatly overemphasized in the etiology and pathogenesis of pure mitral regurgitation. In 66 per cent of our operated series of patients with pure mitral regurgitation, clinically very severe in most cases and with fantastically enlarged left atria in several, there was no detectable enlargement of the mitral annulus. Thus, in this substantial majority of patients, circumferential purse-string constriction carried out blindly or even annular shortening carried out under direct vision, for that matter, would produce stenosis and fail to correct the insufficiency. In these individuals with insufficiency due primarily to loss of substance, the insertion of an artificial prosthesis or artificial leaflet has been the obvious solution for obtaining a good result.

The two basic operative techniques which have been developed for correction of insufficiency, and which we described before this group last year, have not changed appreciably except for refinements based upon greater experience. The mitral valve is visualized by total cardiopulmonary bypass. The standard right posterolateral thoracotomy with the patient in a straight lateral position gives the best exposure to the left atrium and mitral valve. The use of cardiac arrest is contraindicated for the same reasons just mentioned. If there is any element of annular dilatation this is corrected by annuloplasty (shortening of the annulus) as shown in this slide. If instead there is found a signficant loss of valve substance (the case in two thirds of our patients), a prosthetic leaflet of plastic has been inserted by direct suture.

As we have seen a greater number of these valves at surgery, we have been impressed with the wide variations in the pathology. For example, it is not uncommon to observe a valve that is incompetent at two or more places but perfectly competent in between. As a result, certain refinements have come about in the design of our prostheses to suit these variations. One innovation that we have found of considerable value has been the use of these artificial mitral leaflets.

(slide) They are formed of an inert silicone plastic and come out of the mold after curing looking like this.

(slide) At the time of surgery, with the left atrium opened, the leaflet is then trimmed and tailored with the scissors to correct completely the leakage in the particular patient without interfering with those portions of the leaflets that are still perfectly competent. By means such as this, it has become possible to obtain complete corrections of even very large regurgitant leaks. We are not satisfied with mere palliation, but rather the goal has become the complete restoration of the valvular function to normal.

Moreover, even although many of the 27 patients operated upon to date have had advanced clinical disability (A.H.A. Functional Class III, IV), the operative risk has been low (15 per cent). Not only have the initial gains been sustained but our follow-up, which dates back to 1956, indicates continued improvement in some patients long after the operative procedure. As an example of this, here is the phonocardiogram taken a few days ago of one of our early patients operated upon for mitral insufficiency by open cardiotomy in 1956 (Case 2, J. Thoracic Surg. 35: 154-191, 1958). (slide) You see a completely normal tracing without any murmurs evident now almost 2 years later. Needless to say, the clinical improvements in such individuals have been dramatic.

DR. EFFLER (Closing).—Although our enthusiasm for cardiac arrest remains high, we do not believe it is always necessary nor desirable in surgery of the mitral valve. For this reason we do not use it as a routine, but only when an incompetent aortic valve permits blood return of such volume that occlusion of the aorta is mandatory. We believe there is a distinct advantage in visualizing the valve within the beating heart in order to assess the extent and location of the regurgitation.

There is a distinct disadvantage to the open operation for mitral insufficiency at this time. While the surgeon performs his valvuloplasty, utilizing total body perfusion, he has no accurate method of assessing the degree of correction until the operation has been completed and the patient is no longer on the pump-oxygenator run. Some valves are so badly distorted that the surgeon has no way of knowing whether he is actually improving the situation until he has closed his cardiotomy incision, re-established the patient's own cardiac mechanism and then made direct pressure recordings within the left heart. This is quite different from the closed operation for mitral stenosis.

Air embolus has not been a problem with us as yet. We believe that this may be due, in part, to the position of the patient and to the transatrial approach. As we visualize it, this approach with the right side slightly elevated places the left ventricle in a dependent position. Of course, it is still possible to trap air within the left atrium or ventricle and for this reason it is our practice to continue the high flow perfusion for a period of about 10 to 12 minutes. If air has been trapped, it should be extruded in that time and will then appear within the coronary vessels and initiate ventricular arrhythmia. Intracoronary air is a nuisance, but its deleterious effects are transient and easily corrected and it does not have the import of cerebral air embolus.

The fate of the annuloplasty is still of considerable concern to me. It is not hard to visualize these sutures tearing through under the unrelenting impact of the beating left heart. Like Dr. Kay, we have used annuloplasty in those patients whose regurgitation was attributable to dilatation of the annulus. We used this type of procedure simply because we had nothing more to offer the patient and were gratified to find appreciable reduction in the left atrial pressures when the procedure was completed. However, it seems logical to hope that the ultimate operation for mitral insufficiency will be along the lines described by Dr. Lillehei, some form of valve replacement with a practical prosthetic valve.

I appreciate very much this opportunity to appear before the Association.

SATISFACTORY SURGICAL CORRECTION OF PECTUS EXCAVATUM DEFORMITY IN CHILDHOOD

A Limited Opportunity

Kenneth J. Welch, M.D. (by invitation), Boston, Mass.

MATERIAL

ONE HUNDRED and thirty-eight children with moderate to severe pectus excavatum deformity have been evaluated, from 1951 to 1958, with regard to the desirability of surgical correction and comprise the basis for this report. Seventy-six patients (53 per cent) were considered suitable candidates for operation. There were 49 boys and 27 girls (Fig. 1). The remaining 62 patients (47 per cent), mostly infants, did not have progressive deformity and gave clear-cut evidence of improvement by the twenty-fourth month.

ETIOLOGY

The etiology of pectus excavatum deformity is not clear in many cases. However, it is possible to analyze a number of these deformities.

Only 8 patients had inherited disease. There is little doubt about a congenital element in this problem, in that the deformity is uniformly present in the 32 mm. embryo. In addition, it is always present at birth in those individuals who remain in the progressive or worsening group.

There is a condition known as "spinomyelodysplasia" with structural damage to lower dorsal somites and associated defects of the vertebral column, ribs, and cartilage segments. These patients have the most severe problems in that they not only develop the usual structural alterations of the thoracic cage in pectus excavatum but have superimposed scoliosis (Fig. 2).

Pectus deformity may be acquired with steady pressure against the soft and unossified gladiolus. The term "Zapatero de Torax" is used in Cuba. This describes the acquired funnel chest that occurs in youngsters in the shoemaking trade where a leather-cutting instrument is braced forcibly against the lower sternum.

From the Pediatric Surgical Service, Boston City Hospital, and the Department of Surgery, Harvard Medical School, Boston Mass.

Read at the Thirty-eighth Annual Meeting of The American Association for Thoracic Surgery at Boston, Mass., May 16-18, 1958.

Another group is explained by relative inspiratory obstruction. This is most striking in the first year of life and, in our experience, this is the group most likely to remedy itself as the individuals gain greater air inlets. This may be a desperate period for the family. Indeed, 2 youngsters required tracheostomy at the height of bacterial pulmonary infections.

There is no abnormality at the costochondral junction. Longitudinal sections through this zone, after decalcification, show no gross or microscopic alterations. As one studies resected cartilaginous segments, by multiple coronal sections, there is often found a disordered architecture and a situation that implies local circulatory or nutritional damage—a chondodystrophy. Certainly one often encounters very bizarre-shaped cartilage segments in the process of resection.

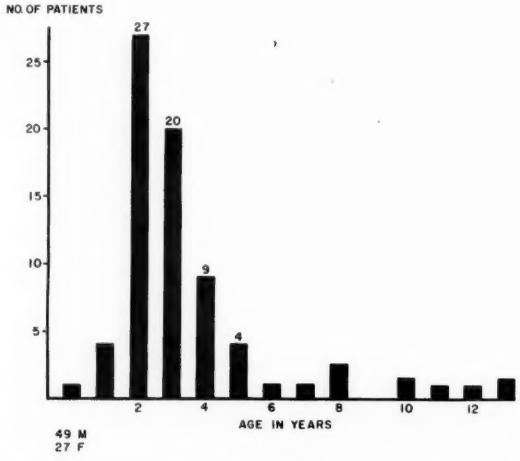

Fig. 1.—Surgical correction of pectus excavatum deformity—76 patients, 1951-1958.

In the group of patients with multiple congenital anomalies, including mental deficiency, there has been an element of hypotonia with poor posture, and the disease may, at times, be the result of complex muscular imbalance.

The concept of a restricting retrosternal ligament, or short central tendon of the diaphragm, has focused attention on the maximal area of deformity and has contributed to the evaluation of this disease.[1] Most writers have accepted this theory and have described fibrous extensions of the linea alba that, when divided, result in a dramatic lift in the central area of the deformity. This has not been confirmed in this series. In the seventy-six operations performed, only loose areolar tissue was encountered behind the xiphoid and there was no significant lifting of the deformity after transection of the xiphoid. It was not until much later in the operation, when all of the deformed cartilage segments had been resected, that one could see a significant structural improvement.

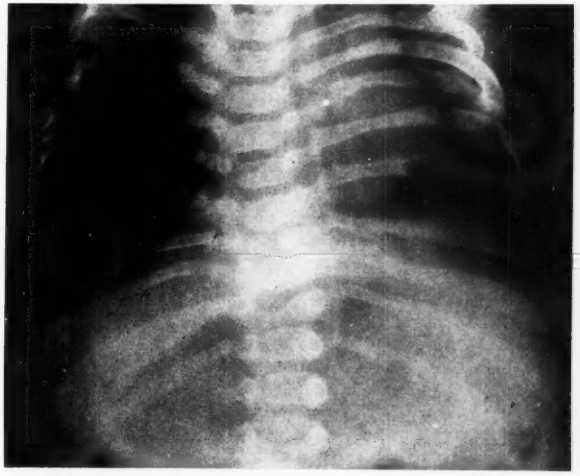

Fig. 2.—Spinomyelodysplasia and pectus excavatum deformity in patient 8 months of age.

Fig. 3.—Typical habitus and poor growth performance in a 3-year-old boy.

SYMPTOMS AND SIGNS

Very few patients were symptomatic at the time of operation; however, all had a history of a difficult first year of life with recurring bronchitis, "croup," and more than the usual number of respiratory infections.

Percentile growth performace was usually in the 50 to 90 per cent range, as plotted on the anthropometric charts of the Children's Medical Center up to the age of 2 years. However, after that we encountered 17 patients who were in the twenty-fifth percentile or below by the end of the fourth year. One Puerto Rican youngster was of unusual interest in that she was hospitalized three times with an x-ray diagnosis of bronchopneumonia and was in the tenth percentile at age 5 years. Two siblings, without this deformity living in the same household, had not had difficulty with respiratory infection and were in the nintieth percentile. The downward drift in over-all physical development with severe funnel-chest deformity in this 2-to-5 age group has become quite impressive (Fig. 3).

Four youngsters complained of chest pain with activity, and 2 had a presenting complaint of intermittent syncope.

Eleven patients had a short systolic murmur at the left border of the sternum; 1 patient had an associated diastolic murmur. This youngster was in the multiple anomaly group and we rejected her as an operative candidate. We have the strong feeling that if there is anything atypical about the murmur in its location, or if it carries a diastolic component, the risk of operation goes up greatly. Such patients should not be operated upon without an anatomic diagnosis of the cardiac lesion by cardiac catheterization.

SELECTION FOR OPERATION

Our position has been arrived at by a trial and error method extending over a 7-year period. In the first period (1951 to 1955), age at the time of operation ranged from 8 months to 14 years. One patient was lost at the age of 16 months—a youngster with multiple congenital anomalies. Shaken by this experience and by the fact that the "limited operation" was unsuccessful in correcting the deformity and required subsequent complete cartilage resection to the angle of Lewis, we have given up operations in the first 2 years of life. Eleven youngsters, more than 6 years of age, were operated upon and the results in this group were not considered to be as satisfactory as in those patients operated upon between the ages of 2 and 5 years.[2] We had greater difficulty with the anesthetic management, with the operative technical procedure, and with the postoperative course. It appears that there is a limited opportunity to obtain complete structural cosmetic and functional correction of this disease.

We like to see all of these youngsters from birth. Lateral chest films should be obtained at each 6-month interval. Two important measurements are made in these lateral films which are taken neither in deep inspiration nor in full expiration. The first measurement is an external skeletal AP diameter of the chest at the angle of Lewis. The second measurement is the distance between

the anterior surface of the vertebral body and the gladiolar-xiphoid junction. The normal ratio between these two measurements is one or greater than one. In mild degrees of pectus excavatum deformity, this drops to between 0.75 and 0.5. In moderate deformity there is a ratio of from 0.5 to 0.3. With severe

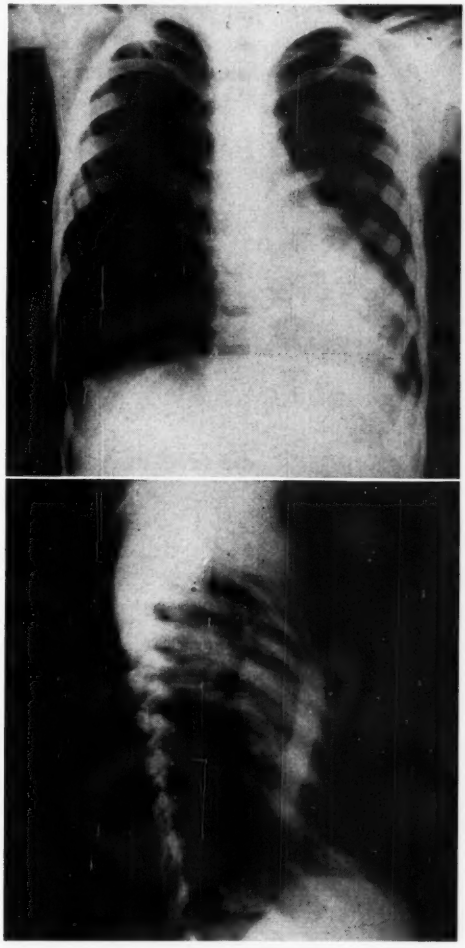

Fig. 4.—Anteroposterior and lateral chest roentgenograms of an 11-year-old boy with severe, irreversible pectus deformity. The sternum is in apparent contact with the vertebral column.

deformity the ratio is 0.3 or below. In several cases, the sternum was in apparent contact with the anterior surface of the vertebral body and a ratio of 0.1 is not unusual (Fig. 4). The significance of this ratio has to be corrected for progressive loss of AP diameter. This occurs in children in the 2- to 5-year age group who, in a matter of months, acquire the typical pectus excavatum

habitus. There is a dorsal kyphos and they slump over the defect. If the youngster's AP diameter is decreasing, and his ratio is constant, we would consider him a candidate for operation with lesser degrees of deformity. In short,

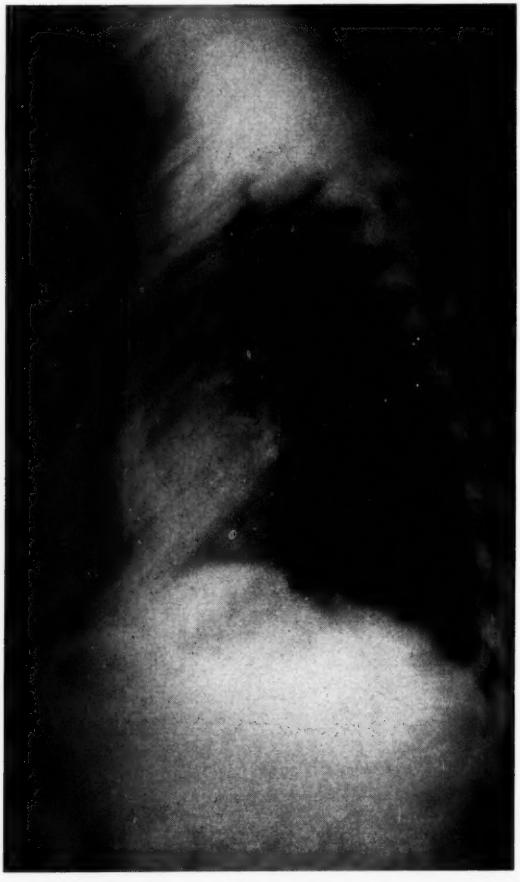

Fig. 5.—Lateral chest roentgenogram of a 2-year-old boy with a normal AP diameter and a measurement ratio of 0.4.

in a youngster with a normal AP diameter, we would like to see evidence of an unimproving ratio of 0.5 or less at the end of the second year before recommending him for operation (Fig. 5). It has not been difficult, on this basis, to sort out the 62 individuals that have not required operative interference. Our estimate of improving cases has been accurate and we have obtained several 6-year follow-ups in this group and found normal chest contours.

PREOPERATIVE STUDIES

Most of the early reports about this disease dealt with the technical aspects of structural correction. In the reports of Ravitch,[3] Dorner[4] Hansen[5] and

others, not all of these individuals went on to enjoy a normal, unhandicapped life with a simple cosmetic deformity. The following routine and special studies were done with the purpose of adding more objective information in this limited age group.

In addition to the height-weight percentile recording and the serial anteroposterior and lateral films of the chest mentioned above, the following additional studies are done before operative intervention is undertaken.

An electrocardiogram is obtained with multiple leads for its diagnostic value and as a base line study for the years ahead. Sixty-three patients had electrocardiograms that were interpreted as normal and 13 had changes associated with displacement of the heart reported as right axis deviation, inversion of T waves, and peaking of P waves. Two patients had bundle branch block.

In the children old enough to cooperate in a Master's type exercise tolerance test, we have entered this into the record, and have repeated this in the postoperative period.

Venous pressure and circulation time was determined in 10 individuals and they were all within the normal range.

Vital capacity and timed vital capacity has been obtained in all youngsters over 2 years of age. Although these are not highly accurate, they give us some reassurance about normalcy in the younger group. This is done with an Emerson-type spirometer and the figures are, with few exceptions, normal for their age and height in comparison with the standards established at the Harvard School of Public Health. They cooperate effectively in this simple study but cannot cooperate adequately for more detailed pulmonary function evaluation (see Special Studies).

We insist that these youngsters not be operated upon if there is any evidence of active upper respiratory tract infection or an abnormal coagulation mechanism.

A left anterior oblique photograph in deep inspiration and expiration is obtained pre- and postoperatively.

SPECIAL STUDIES

A. *Cardiac Catheterization.*—We feel that children with a diastolic murmur should be catheterized before operative intervention. The only patient with a diastolic murmur in this group had multiple congenital anomalies and was rejected as a candidate for operation. Five children, aged 2, 4, 6, 8, and 10, were catheterized preoperatively to determine if: (1) there was any demonstrable cardiac handicap in this age group; (2) if such a handicap was disclosed, how it related to the age of the patient and the severity of his deformity; and (3) would it be possible to improve abnormal preoperative cardiac catheter figures by operation.

Fabricius, Davidsen, and Hansen[6] have reported on cardiac function in funnel-chest deformity in 26 patients as determined by cardiac catheterization. Five of these patients were 14 years of age or under and 2 were 7 years of age,

corresponding to patients within the age limits of our study. Combining these studies: 9 of the children had normal pulmonary capillary, pulmonary artery, right ventricle and right atrial pressures with a normal cardiac output. One patient, a 14-year-old boy with only 1 cm. between the vertebral bodies and the posterior border of the sternum, had a slightly elevated right atrial pressure and a ventricular pressure curve similar to that seen in constrictive pericarditis (a diastolic plateau and dip). It is interesting that the pulmonary artery pressure was not elevated in any of the 10 children studied. It would seem that the neurocirculatory difficulties these youngsters have might be related to interference with cardiac filling. An impedance in the right outflow tract or an elevated pulmonary arterial pressure has not been demonstrated in this age group. It is quite possible that abnormal figures might be recorded in the spinomyelodysplasia group where funnel-chest deformity and structural scoliosis coexist. All patients showed a normal A-V difference, none were desaturated, and none showed evidence of auricular fibrillation in the monitoring electrocardiogram. It can be said, in summary, then, that the occasional patient with abnormal cardiac catheter findings will be encountered at a much later date and probably in the third decade of life.

B. *Pulmonary Function Studies.*—Vital capacity and timed vital capacities can be determined in any youngster 2 years of age or over. Definitive pulmonary function studies require a good deal of cooperation and, up to the present time, have not been made available in a well-standardized way for patients under the age of 5. Nine children have had detailed pulmonary function studies. Seven of these were completely normal or, if they had a measurable deficit, it was of minor degree in the range of 25 to 30 per cent below normal, quite obviously within the limit of error of a test that depends, to a large extent, on the motivation of the child.

Two patients with significantly abnormal figures were older and one could predict, from their general appearance and from their x-ray studies, that they had a reduced thoracic volume and that their figures would be well below the normal range on a height-weight basis. The deficits in these 2 individuals were great enough to make them unable to participate in vigorous activities, but they were completely comfortable at rest and with ordinary daily events (Table I).

The discouraging finding was that 1 patient had preoperative studies at the age of 8, was found to be 56 per cent of normal, and 2 years after operation was 54 per cent of normal. The second patient, studied preoperatively at age 11, was 65 per cent of normal, and at age 14, 3 years after operation, was 52 per cent of normal. There was no desaturation and there was normal gas transfer. The deficit appeared as one might expect in vital capacity, total lung capacity, and maximum breathing capacity. Table II summarizes the abnormal findings in these 2 patients compared with 2 younger patients in whom it was possible to carry out a satisfactory correction of the deformity. A report from the Oresundhospital, Copenhagen, by Hansen and Jacoby,[5] indicates that with

TABLE I. PREOPERATIVE STUDY OF PULMONARY FUNCTION IN SEVERE FUNNEL-CHEST DEFORMITY

R.D.—10 YR.		% OF NORMAL
Maximum breathing capacity (L./min.)	67	78
Vital capacity (c.c.)	1,720	64
Timed vital capacity		
1 sec. % total	79	
2 sec. % total	94	
3 sec. % total	99	
Walking ventilation (L./M.²/BSA)	15.8	
Total lung volume (c.c.)	2,300	65
Residual volume (c.c.)	640	
Residual volume/total capacity (%)	28	
Resting minute ventilation (c.c.)	8,955	
Resting minute O_2 uptake (c.c.)	231	
Ventilation equivalent	3.9	
Pulmonary mixing index (% N_2)	1.68	
Breathing reserve	86.6	

severe deformity and a *severe* pulmonary function handicap, there may be significant improvement following operation. They report 4 patients in whom differential bronchospirometry was carried out in the age range of 18 to 27 years. The function in the two lungs was identical. This is surprising in view of the fact that the heart is grossly displaced into the left hemithorax. Combining the performance of the two lungs in order to compare them with the figures reported here, again there was no difficulty in the mechanics of respiration or in diffusion. The deficit proved to be in vital capacity, total lung capacity, and in maximum breathing capacity. One of the patients is reported in detail. His preoperative vital capacity, both lungs combined, was only 430 c.c., the total lung capacity was 850 c.c., and the maximum breathing capacity 12 L. Postoperatively, this increased to 1,400 c.c. for vital capacity, 1,900 c.c. total lung capacity, and 22.4 L. maximum breathing capacity. Fluoroscopically, the change appeared to be principally in the elimination of paradox. Here then we have an individual who, postoperatively, has only 25 per cent of normal lung function yet, by comparison with the preoperative status, was vastly improved.

TABLE II. PULMONARY FUNCTION STUDIES IN PECTUS EXCAVATUM DEFORMITY*

PATIENT	AGE IN YEARS	TOTAL VOLUME CAPACITY (1-3")	VITAL CAPACITY (C.C.)	TOTAL LUNG CAPACITY (C.C.)	MAXIMUM BREATHING CAPACITY (L./MIN.)
G. R.	3	N	600	----	
	6	N	1,150	1,837	35
N. P.	6	N	2,150	----	43
	8	N	3,120	3,680	74
H. H.	8	N	1,850 (56%)	----	--
	10	N	1,750 (54%)	----	61
R. D.	11	N	1,720 (64%)	2,300 (65%)	67
	14	N	2,100 (52%)	2 800 (56%)	84

*Significantly low pulmonary function figures (vital capacity, total lung capacity, and maximum breathing capacity) are not returned to normal by operation in the older child with "irreversible deformity."

In summary, then, in 7 of the patients studied, there was no evidence of pulmonary insufficiency. In 2, the figures are suggestive of impending pulmonary insufficiency. Complete and permanent structural correction of the

individual over 8 years of age with severe funnel-chest deformity is only rarely accomplished. The center of the chest is raised but the declination of posterior rib segments remains and thoracic volume is not increased. If there is a pulmonary function deficit demonstrated preoperatively, there is little or no improvement in these figures postoperatively.

Looking ahead, one cannot predict with certainty the clinical course of any child with marked pectus excavatum deformity that remains uncorrected. Some, to be sure, will develop paroxysmal fibrillation, precordial or radiating pain, syncope, low blood pressure, dyspnea at rest or on exertion, cardiac failure and/or pulmonary suppuration.

MULTIPLE CONGENITAL ANOMALIES

Six patients had multiple congenital anomalies. Having had the bitter experience of losing one of these patients and finding reports of others,[7] multiple anomalies might be suggested as a categorical contraindication to operative correction of pectus excavatum deformity. In 1 case there was coexisting Marfan's syndrome with a dissecting aneurysm of the thoracic aorta. This burst in the immediate postoperative period. In our patient, a 16-month-old child, there was an associated cleft palate deformity which had been successfully corrected, scoliosis, pectus excavatum deformity (severe), and poor central nervous system development. Another patient, 2 years of age, with Marfan's disease and congenital absence of the pectoral muscles on the right, was rejected for operation. The third patient was a 2-year-old with Klippel-Feil syndrome, ovarian agenesis, severe pectus excavatum deformity, and an element of mental retardation. The fourth had multiple hemivertebrae, absence of costal cartilages, D_3 to D_8, a deficiency of intercostal muscles, a diastolic and systolic murmur, and severe pectus excavatum deformity. She was rejected for operation because of the complex murmur and the multiple associated anomalies. The fifth, 7 years of age, had bilateral microtia, mental deficiency, and a roaring Roger-type murmur. The sixth and oldest patient, 10 years of age, had scoliosis, deficiency of abdominal muscles, bronchiectasis, and pectus excavatum deformity. This probably again represents spinomyelodysplasia. His bronchiectasis is arrested and he has been free of pulmonary infection for some time.

Continuing to operate upon this group will result in excessive postoperative morbidity and/or mortality. It will add unnecessary deaths in connection with the surgical management of pectus excavatum deformity and will discredit the procedure.

OPERATIVE MANAGEMENT

It is seen in Table III that the first thirty-four operations were performed after the method of Sweet.[8] Parallel chondrotomies are carried out in two rows with anterior cortex sternotomy, elevation of the sternum, and reconstruction of the chest wall, using the elevated sternum as the keystone of an arch. The cut cartilage ends are accurately resutured. This seemed appealing because, upon

completing reconstruction of the chest wall, there was a stable situation. These patients seemed to have little flail motion when they were allowed to resume their own breathing cycle. In 4 patients we extended this operation to include the costal complex and there was less effective breathing postoperatively. The costal arch plays a very important role in diaphragmatic excursion.

TABLE III. TYPE OF OPERATION PERFORMED

1. Multiple chondrotomies with suture fixation (Sweet)	34
2. Subperichondrial resection with transverse sternotomy (Garnier, Lester, Ravitch)	39
3. Xiphoidectomy and retrosternal freeing (Brown, Lester)	2
4. Cartilage resection with rib strut (Dailey)	1
Total	76

There was a great deal of blood loss with this procedure. We were not able to stay out of both pleural cavities in any case so that we had, by definition, the postoperative problems of bilateral thoracotomy. These youngsters had occasional atelectasis, they had to be carefully checked for tension difficulties, and many developed pleural cavity collections of blood and/or serum that required tapping. We were fortunate not to have mediastinal or pleural cavity infection in this group. As we gradually arrived at the time of election for operation, we found that a better immediate and permanent correction and a less hazardous one could be obtained using elements of the operations described by Garnier,[9] Lester,[10] and Ravitch.[11]

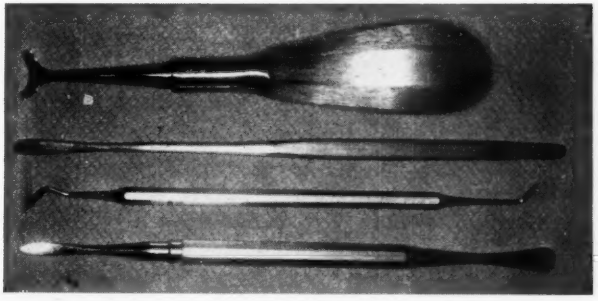

Fig. 6.—Dental instruments are ideally suited for performing costal cartilage resections in the younger age group.

Our operative procedure can be described briefly. We use a short, transverse skin incision over the maximal point of the deformity. It is not necessary to carry this beyond the nipple line. Subcutaneous tissue dissection is avoided. The pectoral muscles are swept off the chest wall and the bony thorax is exposed from the angle of Lewis to the costochondral junction on each side from the third to the eighth ribs. The xiphoid is grasped with an Allis clamp and is carefully cut through at the gladiolar junction. A technical innovation that we have arrived at is to make T-shaped incisions with the short arm of the "T" at the sternal border of the costal cartilage, extending the long arm out to the costochondral junction. The cartilage envelope is opened medially, using dental instruments (Fig. 6). We have found that the usual rib resection instruments

are inadequate for this maneuver, and the younger the patient, the more this is true. It is possible, and has been possible in the last 24 patients, to carry out symmetrical cartilage resections on each side without entering either pleural cavity and without injuring the internal mammary vessels. It is possible to clear the cartilage medially for about three quarters of its circumference, permitting it to be grasped with an Allis clamp. Then, as upward pressure is exerted, using a fine blade, the cartilage is divided at its sternal insertion. The cartilage end lifts up. It is then possible to encircle the costal cartilage

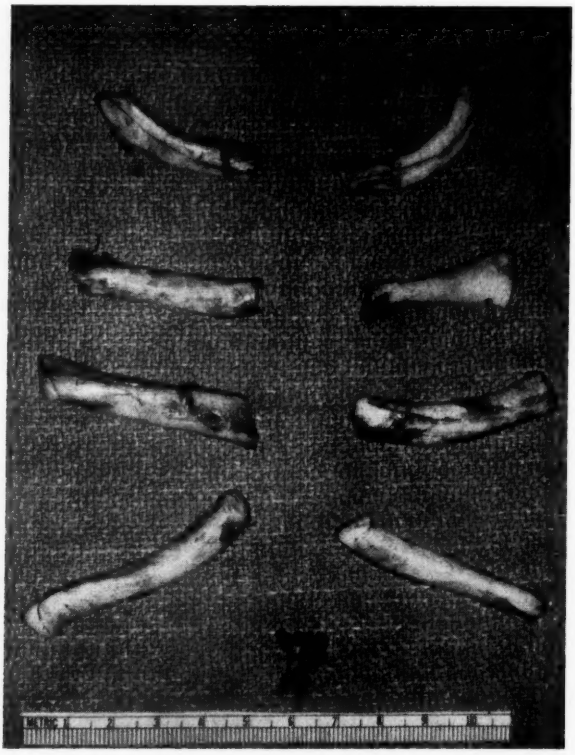

Fig. 7.—Resected costal cartilage segments, xiphoid, and a wedge of gladiolar cortex.

and gain access to a dry, blue-gray periosteal envelope and to quickly shell out these cartilages, breaking them off at the costochondral junction (Fig. 7). We have gone beyond the costochondral junction in a few cases where it did not seem to include the lateral extent of the deformity. The recti are not swept off the costal arch. The intercostal bundles are not divided. An anterior cortex sternotomy is performed, creating a broad V-shaped trough. The sternum can then be drawn forward, fracturing the posterior cortex. It is held in this new position using 1-0 silk, and the periosteum is reinforced with 4-0 silk. The muscles are reapplied to the chest wall with 3-0 silk. We are careful

to obliterate dead space by suturing the pectorals to the intercostal muscle groups and to the periosteum of the sternum. One other technical point is that we then run off the inverted Y-shaped silk closure of the pectorals and the rectus muscle with 3-0 chromic catgut, making this a water-tight situation and separating it from the superficial layers of the wound. There follows a subcuticular build-up using fine catgut with interrupted 5-0 silk to the skin.

We do not carry out an extensive mediastinal dissection with the finger. We have passed a finger behind the sternum, in the midline in each case, to be sure that there are no remaining restricting elements. However, in none of these patients have we encountered anything but loose areolar tissue.

An elastoplast dressing is applied with no protective support and no type of external traction is used.

Gravimetric estimates of blood loss has been as low as 155 c.c. and averages about 350 c.c. in this 2- to 5-year age group. Operative time has been in the range of 1½ to 2 hours. This seems important because of the great responsibility that the anesthetist has in this operation and fatigue is a function of time in monitoring this complex respiratory situation. It also decreases the amount of whole blood replacement necessary and adds substantially to the smoothness of the postoperative course.

ANESTHETIC MANAGEMENT

Anesthetic management is by far the most difficult and most responsible aspect of the operation in this age group. It is, at best, a tricky anesthesia and, at its worst, a downright dangerous one. The two deaths that we report have both been in connection with anesthetic management. In one, the problem was a complex one because of multiple congenital anomalies. In the other, a 4-year-old boy, there was a clear-cut cardiac arrest on the basis of airway anoxia that occurred as the skin sutures were being placed after a 1½-hour extrapleural procedure with minimal blood loss.

One problem in this closed, temporarily flail chest is in keeping the median pressure in the anesthetic system in an atmospheric range. It does not seem possible to accomplish this accurately with the automatic breathing devices that have recently come on the market. If this can be kept a short procedure and if fatigue is not to enter in, it would still seem best to manage these patients with controlled respiration and manual control of the bag. It may be necessary, in order to keep the lungs inflated, to have occasional pressures as high as 100 cm. of water. If continued, this may so interfere with cardiac filling that you precipitate a cycle of difficulties in this respect. We have asked the anesthetist to place a stethoscope at each base and to watch vital signs and blood replacement with extreme care. It is largely a matter of experience with the manual control of breathing and a sense of adequate ventilation at the alveolar level. One of the problems here is that the anesthetist cannot see the lung—that he has a closed and not an open chest. On the one hand, there is failure to accomplish adequate ventilation at the alveolar level and, on the other, the danger

of interfering with cardiac filling by overdistention of the lungs. The postoperative sequelae of vigorous overdistention of the lungs for any period is well known and has been encountered twice. This sets the stage for ineffective ventilation, atelectasis, and cardiac failure.

Some of these youngsters have developed twitching, some have had unexplained periods of desaturation, others have had hyperthermia during the course of anesthesia.[12]

It must be stressed that anesthetic management is the most dangerous aspect of the surgical correction of this disease and that one must be very detailed in the preoperative evaluation of these patients.

COMPLICATIONS

The most frequent postoperative complication (Table IV) was recurrence of the deformity. This occurred in 7 patients, if one considers the continued progression of the deformity after xiphoidectomy alone carried out in 2 patients in the first year of life. The five true postoperative recurrences occurred in the first part of the series using the Sweet technique. Four of them occurred in youngsters over 6 years of age. Two of these were severe enough to justify correction. One was done by subperichondrial resection with external traction for a period of 2 weeks. The other has not been operated upon.

TABLE IV. POSTOPERATIVE COMPLICATIONS

Recurrence of deformity	5
Keloid formation	4
Marginal necrosis of skin flap	4
Superficial wound infection	3
Chondritis	2
Cardiac arrest	1
Cardiac failure	1

The thing that has confused us about recurrence of the deformity is that it does not develop early and does not relate to whether one does or does not use external skeletal traction. None of the reported internal fixation or external traction methods are continued for more than a month, and most of them are discontinued at the end of the period of hospitalization—roughly 10 days to 2 weeks. The patients with recurrence all look satisfactory at the end of the first month and then, much to one's surprise, after 6 months or a year or 2 years, these youngsters come back with a reacquired funnel. In spite of this, their over-all improvement is satisfactory, they have lost their paradox, and they go on to acquire adequate AP diameter unless they had a markedly decreased AP diameter at the time of operation. The rib strut operation suggested by Dailey,[13] has been tried in only 1 patient. This would seem to be a worth-while maneuver in the older child with recurrence. We have had no experience with the Kirschner wire support of these deformities.[14]

Keloid formation related directly to the use of the vertical incision; this was used eighteen times. Marginal necrosis of the skin flap occurred either with the long vertical incision or in the few patients that we attempted an inverted

"U" incision. The apex of these flaps sloughed without exception. We then shifted to a transverse incision with a slight convexity upward, extending out to the nipple lines and encountered no more keloids or marginal sloughs. Superficial wound infection occurred in 3. Chondritis occurred in 2—1 of these had to be reoperated upon for removal of multiple parasternal silk sutures. The two deaths in this series, listed here as cardiac failure and cardiac arrest, are reported above.

The great majority of patients operated upon at the ideal age, using an extrapleural procedure, have a completely uneventful postoperative course, are allowed to be up and around after the first day, and are discharged home without a dressing on the seventh day (Fig. 8).

Fig. 8.—A 28-month-old boy on his first postoperative day. Cardiac catheterization and pulmonary function figures were normal.

FOLLOW-UP

The follow-up period extends over a 7-year period from 1951 to 1958. In 1954 and 1955, we were operating upon nearly every youngster that presented a moderate to severe deformity. With disappointments in the older group, with recognition of the risk in the multiple congenital anomaly patient, with a better understanding of operative and anesthetic management, and with better experience in predicting spontaneous improvement, we have arrived at the policy of management described above. We do not now operate upon any youngster under 2 years of age. If one accepts this criterion alone, more than half of the patients will drift through to normalcy without operative intervention.

Eight patients have been followed less than 1 year; 10 patients, 1 year; 17, 2 years; 10, 3 years; 15, 4 years; 9, 5 years; and 7 patients have been followed 6 years or more.

Three of seven recurrences have been corrected surgically. Three are of a minor degree and one is a complete failure but secondary operation has been refused.

The results in 67 patients are considered to be excellent.

SUMMARY

1. We are satisfied with the long-term cosmetic and functional result following an extrapleural, subperichondrial resection operation, without external support, performed on selected individuals in the 2- to 5-year age group with pectus excavatum deformity.

2. Two of 76 patients died in an attempt to correct this deformity. Seven patients developed some degree of recurrence and therefore were considered to be unsatisfactory even though subsequently corrected by a second operation. An excellent result was achieved in 67 patients.

3. Studies of cardiopulmonary function are reported. These indicate that within the ideal elective period for operation, 2 to 5 years, there is no objective evidence of abnormal function. There is no evidence of pulmonary insufficiency until the volume of the thorax is grossly reduced below normal in the first decade of life. Two individuals had reduction in pulmonary function to the 50 per cent level. They were not returned to normal levels by elevation of the sternum. Surgical correction at a later age, although cosmetically partially effective, does not convert the habitus of these individuals to normal. When there is objective evidence of a cardiopulmonary handicap, they may be improved from severe to better but are not returned to a normal range.

4. Because of the inability to predict the future course of children with marked, progressive pectus excavatum deformity, they should be recommended for operation by the third year of life if they are to be restored to normalcy in all cases.

The pulmonary function and cardiac catheterization studies were performed by the Cardiopulmonary Laboratories of the Harvard School of Public Health, the Children's Medical Center (Sharon Cardiovascular Unit), the Boston Floating Hospital, and the Boston City Hospital.

A number of these patients were operated upon at the United States Naval Hospital, Chelsea, Mass., under the direction of Captain Louis Haynes. I am indebted to him for the able assistance and encouragement given during the early part of this study.

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(For Discussion, see page 724)

PECTUS EXCAVATUM: AN APPRAISAL OF SURGICAL TREATMENT

Paul C. Adkins, M.D. (by invitation), and Owen Gwathmey, M.D., Washington, D. C.

IT IS NOW well established that surgical correction of a funnel chest deformity is feasible and relatively safe. There is, however, little agreement in the literature concerning age limitations, if any, for surgical correction, and even less unanimity of opinion about the actual surgical technique to be used. The purpose of this presentation is to review our experiences in the correction of pectus excavatum deformities over the past 5 years. Our present approach to this problem is one which has been achieved by a process of trial and error. The operation is best performed during childhood and correction of these deformities is usually less formidable at this time than in later years. However, our experiences have led us to believe that with our present techniques, correction of a pectus excavatum may be carried out on teenagers and young adults with equally good results.

We have little to add in the realm of speculation concerning etiology of this deformity. Claim has been made that a hypertrophied or contracted substernal ligament may be the etiological agent.[3, 11, 14, 17] Although some attachment of the undersurface of the sternum to the diaphragm may be readily apparent when this deformity is seen in infancy,[12, 13] we are not sure that we have ever encountered a hypertrophied substernal ligament in our surgical experience. Once past infancy, however, there appears to be considerable variation in the amount of progression of this deformity. Following children over periods of several years we have found some sternal depressions to become more pronounced, others remain static, while others may become even less apparent. Thus far we have been completely incapable of predicting the natural progression of any of these deformities.

INDICATIONS FOR OPERATION

The prevention or correction of cardiorespiratory embarrassment is considered a prime indication for operative correction of a pectus excavatum. There can be little question that reduction in the anterior-posterior diameter of the mediastinum, resulting from sternal depression, can cause distortion or rotation of the heart and great vessels.[2, 7, 16, 20] The cough mechanism is often

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impaired as a result of this deformity.[4] Although, cardiorespiratory problems are relatively unusual during childhood,[9, 20] progression of the deformity may result in later difficulty.[16] The other and equally valid indication for surgical correction is for cosmetic and psychologic reasons. A depression or deformity of the chest wall may be a source of considerable embarrassment, especially during adolescence and young adulthood. Consequently, operative correction of such a deformity is well justified even in the absence of symptoms.

SYMPTOMS

It is our impression that the symptoms associated with a pectus excavatum are probably more frequent than is generally appreciated.[15] Although patients or the parents of the patients with a pectus excavatum frequently disclaim any limitation of activity, closer questioning often reveals that such patients have been slightly below normal in their activities when compared with their contemporaries. Many such patients have had rather frequent and prolonged respiratory infections. Six of the children whom we have seen have been treated by their pediatrician for varying periods of time for so-called "allergic bronchitis." The five adults in this series have all had some degree of disability associated with their deformity. In each instance, the disability consisted of moderate to severe exertional dyspnea and fatigability.

CLINICAL MATERIAL

Thirty-nine patients with a pectus excavatum have been seen at The George Washington University Hospital in the 5-year period ending in October, 1957. Five patients were considered to have a minimal deformity with no associated symptoms and no operation was advised. Six small children were believed to be candidates for operation, but surgical intervention has been deferred until they are older. One adult patient with a rather marked deformity and associated symptoms refused operation. The remaining 27 patients have been subjected to a total of twenty-nine operations for the correction of a pectus excavatum deformity. There were, in the entire group of patients seen, 24 males and 15 females. In the patients who underwent operation, the age range was from 3 months to 34 years (Table I). Four of these patients were adults. Two of the adults showed associated manifestations of Marfan's syndrome.

TABLE I. PECTUS EXCAVATUM
AGE DISTRIBUTION AT TIME OF OPERATION

AGE	NO. OF PATIENTS
3 mo.-18 mo.	5
18 mo.-3 yr.	4
3 yr.-6 yr.	6
6 yr.-16 yr.	8
Over 16 yr.	4
Total	27

SELECTION OF OPERATION

There is little doubt that the simple operation described by Brown[3, 4] (excision of the xiphoid and division of the substernal ligament) can be carried

out with a high degree of success if performed prior to the age of 1 year (Fig. 1). The operation is brief, relatively atraumatic, and a satisfactory cosmetic result may be anticipated, particularly if there is some degree of paradoxical motion of the depressed portion of the sternum. In our hands in 5 cases, the operation has yielded a good to excellent cosmetic result in four instances (see Table III). In the fifth case, an 18-month-old female, the initial operation produced virtually no cosmetic improvement, consequently, a subsequent, more radical operation with resection of the costal cartilages was performed at the age of 3½ years. Thus, even though the maximum cosmetic result may not be achieved from this simple operation of Brown, it may make a second operation at a later date less formidable.

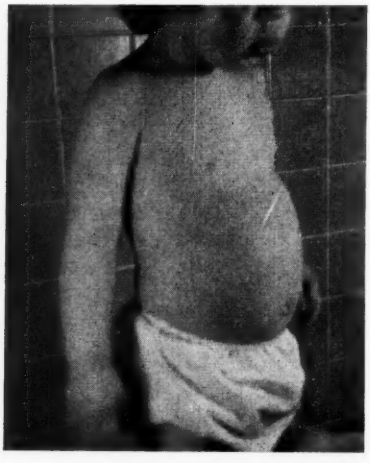
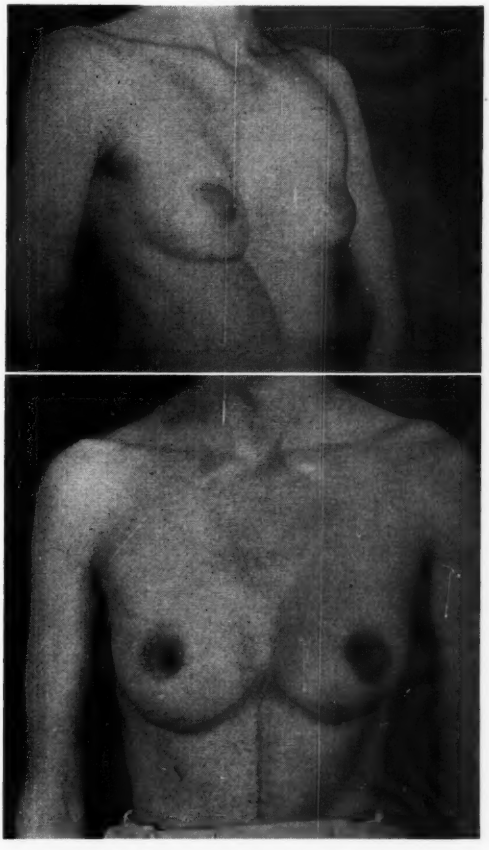


Fig. 1.—Photograph of a 2-year-old child, 18 months after simple division of substernal ligament. No residual sternal depression.

It is presently our policy to defer operation, if at all possible, on patients between the ages of 1 and 3 years. During this age, the expected response to the simple operation of Brown is not satisfactory.[9] At the same time, these children are not considered ideal candidates for the more radical operation of chest wall reconstruction. Although they can tolerate the surgical procedure itself fairly well, they are unable to cooperate in the immediate postoperative period. They do not cough, and a tracheostomy may be necessary. The one death in this series was in a child of 2½ years of age who developed an acute bilateral pneumonia 24 hours after operation. She had a history of frequent severe respiratory infections and allergic bronchitis. She was considerably undeveloped for her age. The child was operated on at this age, with some misgivings, in the hope that the correction of a severe pectus excavatum deformity might alleviate some of her respiratory problems and increase her growth. Although the operation was uneventful, this child developed respiratory difficulty approximately 12 hours postoperatively and pursued a rapid downhill course. The 3 other children operated on in this age group have also been more

difficult to manage postoperatively than the older children. We now prefer to wait until these children have reached a minimum age of 3 years, both mentally and physically.

A.



B.

Fig. 2.—*A*, Preoperative photograph of a 29-year-old women with marked pectus excavatum. This patient had moderate exertional dyspnea.
B, Postoperative photograph demonstrating submammary scar with downward extension.

In patients over the age of 3 years, some form of plastic reconstruction of the anterior chest wall has been employed. The principles of such an operation are:

1. Adequate exposure of the depressed portion of the sternum and adjacent deformed costal cartilages.

2. Freeing of the undersurface of the sternum from its ligamentous and fascial attachments to the mediastinum and diaphragm.

3. Resection of the involved costal cartilages, including division of the attachments of the rectus muscles to the costal margins when deemed advisable.

4. Osteotomy of the sternum. This may be either a transverse osteotomy or "T" fracture depending upon the severity of the deformity and the ease of adequate mobilization of the sternum (Fig. 3).

All operations have been performed under general endotracheal anesthesia. We currently favor the bilateral submammary incision for several reasons. It yields a far better cosmetic result, especially in females, while affording excellent exposure of the lower sternum and adjacent costal cartilages. In addition, it gives easy access to the costal margins, which often flare and need tailoring. This type of transverse incision does not overlie the osteotomy of the sternum, hence a direct bone marrow to skin communication is avoided.

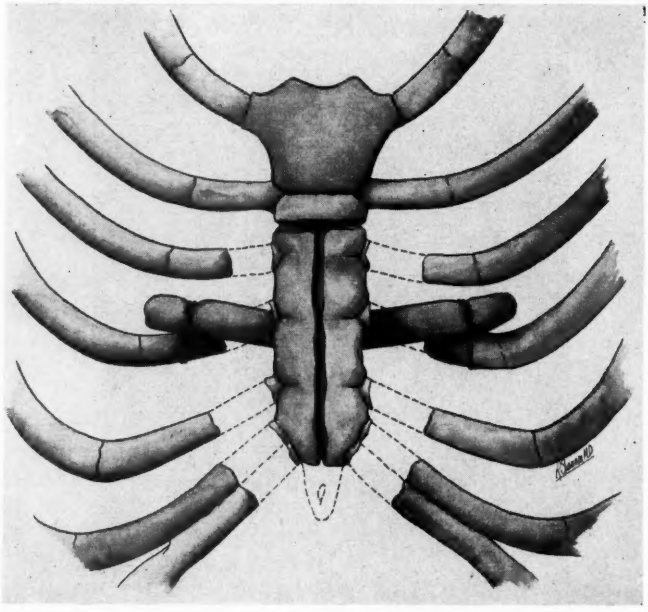
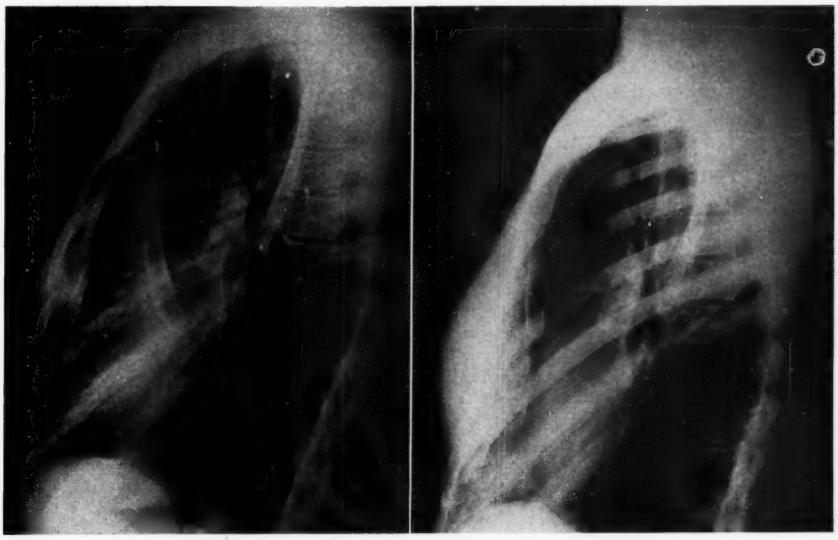


Fig. 3.—Diagram showing homologous rib graft anchored in place and one type of osteotomy of the sternum. The rib strut is also anchored to the pectoral muscles (not shown).

We do not reflect the pectoralis muscles off the rib cage. These muscles can be split in the direction of their fibers to expose each cartilage to be resected. The muscles can be then reapproximated with a few interrupted sutures. We believe that this avoids a considerable amount of dissection, unnecessary trauma, and blood loss. It does not interfere with the over-all cosmetic result.

A variety of techniques have been described for maintaining the sternum in an elevated position once it has been fully mobilized.[1, 2, 5, 6, 8, 9, 10, 13, 17, 18, 19, 21] We have tried a number of these techniques, including forms of external traction, resuturing the costal cartilages and sternum together after wedge osteotomy,



A. *B.*

Fig. 4.—*A*, Preoperative roentgenogram of same patient as Fig. 2, *A*.
B, Postoperative roentgenogram after chest wall reconstruction using homologous rib as a strut.

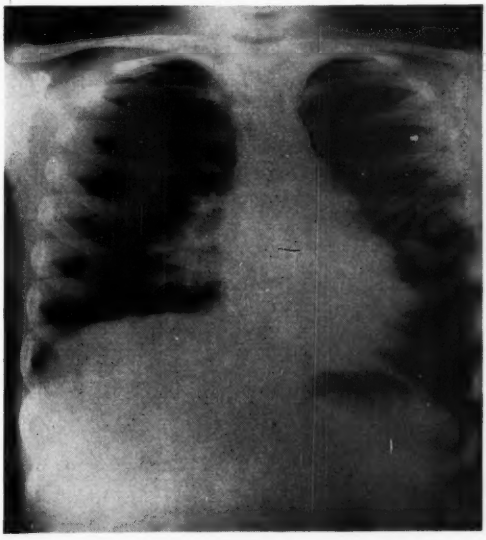


Fig. 5.—Postoperative roentgenogram showing use of homologous rib strut in a 3-year-old child. Good cosmetic result.

and the use of a homologous rib graft as a strut. The technique of passing a homologous rib graft beneath the sternum and anchoring it to the appropriate costal cartilages was first advocated by Dailey in 1950.[5, 6] We started using this technique on adults and teenagers with severe sternal depression. It was found to be so satisfactory that the use of such a strut has now been extended to the younger age group. A strut has been used on 4 children under 6 years of age in our series.

We believe this technique has several advantages over other methods. No form of external traction device is necessary, obviating the need for cumbersome appliances on the chest wall. The strut gives stability to the sternum

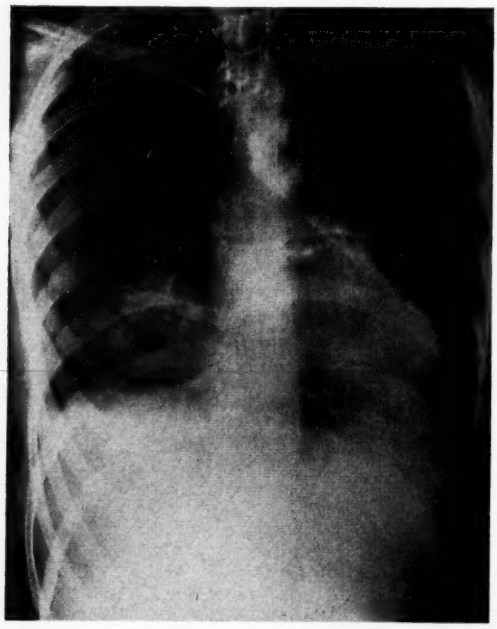


Fig. 6.—Homologous rib strut postoperatively in a 23-year-old patient. Note notch cut in strut.

and anterior chest wall, preventing paradoxical motion in the postoperative period. The sternum is readily held in a slightly overcorrected position, a situation which we believe gives a better cosmetic result in long-term follow-ups, particularly in those children who are in a period of rapid growth. It has been our experience to perform operations without the use of a strut, and with an apparently good cosmetic result at the conclusion of the operation. We were then disappointed and chagrined to see these patients a year or 18 months later with some degree of recurrence of their sternal depression. This has not happened in any case in which a strut has been used; some patients having been followed 5 years or more.

A strut has been utilized in the correction of a pectus excavatum in 14 patients. For the most part we have used a homologous rib graft as the strut.

These ribs are removed from patients undergoing thoracotomy, who are free from infectious or malignant diseases. The ribs are then cleaned thoroughly and preserved in a deep-freeze in sterile containers. In three instances we have used synthetic struts made of a methylmethacrylate resin. These were fashioned at the time of operation, but proved to be rather cumbersome and were accompanied by an excessive amount of tissue reaction in the immediate postoperative period.

The major disadvantage to the use of a strut is the possibility of infection. This has occurred in 4 cases, as a persistent draining sinus postoperatively. In each case, the strut was removed easily, 1 to 4 months after the initial operation. In only one instance was there any loss of cosmetic result. This was in a 19-year-old male with Marfan's syndrome. After the removal of the strut in this case, the sternal depression was again present, but to a much lesser degree than preoperatively. Those patients in whom a strut was used but not removed, showed gradual absorption of the homologous bone over a period of 6 to 8 months.

The other disadvantage to the use of a homologous rib graft has been the inability to obtain enough variation in size and curvature of the graft to meet the needs of the individual patient. To avoid this and to attempt to circumvent the problem of infection, we are currently working on the use of a thin, flat, stainless steel bar. This bar has several notches on either side to facilitate anchoring to the costal cartilages, and a hole at either end to make removal easy. It is proposed to use these bars as struts, and remove them electively after complete healing of the chest wall has occurred.

POSTOPERATIVE MANAGEMENT

The occurrence of a unilateral or bilateral pneumothorax during these operations has not been a problem. This has occurred ten times in twenty-nine operations, including five instances in children under the age of 6 years. In 4 cases the pleura was inadvertently entered bilaterally. If penetration into the pleural cavity is recognized at the time of operation, an intercostal catheter for water-seal drainage is inserted at the conclusion of the procedure. In any event, a portable chest roentgenogram is taken in the operating room at the conclusion of each operation. If a previously unrecognized pneumothorax is seen at this time, it is a simple matter to insert an intercostal catheter since the patient is invariably still asleep. No untoward effects have resulted from the creation of a pneumothorax. Chest catheters can almost always be removed within 24 hours, as soon as there is radiologic evidence of full re-expansion of the lung.

The major problem in the immediate postoperative period has been to keep a clear tracheobronchial tree. This is particularly true in the younger children during the first 48 hours after operation. The patient is usually allowed out of bed by the fourth or fifth postoperative day, and can leave the hospital by the eighth or ninth day.

There is another important aspect of the postoperative care which we do not believe has received sufficient attention in the literature. This is the problem of postural training and over-all muscular balance. It has been our experience to perform what was apparently a successful correction of a funnel chest deformity, only to find that the patient was still potbellied, stoop-shouldered, and kyphotic. Any significant degree of sternal depression, which has been present for a period of years, is accompanied by a weakness and shortening of the pectoral, trapezius, and abdominal muscles and, to some degree, the other accessory muscles of respiration. Stretching and strengthening of these involved muscles, as well as postural training, plays an important role in the achievement of a successful cosmetic result. Exercises to improve the strength of these muscles and to regain their normal length can be taught to children as young as 4 years of age, and can be supervised by parents at home. These exercises and postural instructions are usually started at the end of the first postoperative week by members of our Department of Physical Medicine. In those children too young, or unable to follow such a program, the course is started as soon as they have reached a suitable age. The continued use of such exercises and training has improved the over-all appearance of the patients. We feel that this is an important adjunct to operation and contributes to a lasting good cosmetic result.

RESULTS OF OPERATION

Objective evaluation of results after operations for pectus excavatum is admittedly somewhat nebulous. Our evaluation of improvement is based on cosmetic appearance 6 months or more after operation. On this basis we have classified results as follows: (1) good, meaning no significant residual sternal depression; (2) fair, some residual depression but improvement over the preoperative status; (3) poor, showing no significant change over preoperative appearance. On this basis, the over-all results after all operations are given in Table II. Thus, twenty-one results could be classified as good, and four as fair. These results include 2 cases in which secondary operations were necessary to improve the cosmetic appearance.

TABLE II. PECTUS EXCAVATUM RESULTS—ALL OPERATIONS

AGE	GOOD	FAIR	POOR	DEAD
3 mo.-18 mo.	4	–	1*	–
18 mo.-3 yr.	2	1	–	1
3 yr.-6 yr.	4	1	1*	–
6 yr.-16 yr.	6	1	1	–
Over 16 yr.	3	1	–	–
Total	19	4	3	1
Final total	(21)		(1)	

*Reoperation with good results.

In 14 patients in whom a homologous rib graft or other strut was used, 12 could be classified as good results (Table III). There was one fair result, and this was in a patient who had the strut removed approximately 1 month postoperatively. One death occurred in this group, as mentioned previously.

In the group in which no strut was used the over-all results were not as gratifying. In the 5 infants subjected to the simple operation of division of the substernal ligament, 4 had good results, and 1 infant required a second operation. In 10 patients who had chest wall reconstruction without employing a rib graft as a strut, the results in 5 cases were fair or poor. Good results were obtained in the remaining 5 cases, including 2 in which an external traction device was used. One of the patients in this group with a poor result has been subjected to a second operation using a homologous rib graft with a good result. One other patient needs a secondary operation, but this has not yet been done.

TABLE III. PECTUS EXCAVATUM
RESULTS OF VARIOUS OPERATIONS

	GOOD	FAIR	POOR	DEAD
Division of ligament	4	-	1	-
Reconstruction—no strut	5	3	2	-
Reconstruction with strut	12	1	-	1
Total	21	4	3 (1*)	1

In terms of subjective symptoms, the 4 adults in this group had all definite symptoms of exertional dyspnea and fatigability prior to operation. In each instance, after correction of the depressed sternum, there has been moderate to marked improvement in their symptoms. In addition, all of these patients have a high morale as the result of their cosmetic improvement.

CONCLUSIONS

1. Surgical correction of a pectus excavatum is feasible in teenagers and young adults, as well as during childhood.

2. Excision of the xiphoid and division of the substernal ligament gives satisfactory results if the operation is performed prior to the age of 1 year.

3. The best cosmetic results in our hands, in both children and adults, have been achieved by the use of a strut passed beneath the sternum.

4. The use of muscular exercises and postural training is an important adjunct to surgery in achieving a good over-all cosmetic result.

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DISCUSSION

(PAPERS BY WELCH [PAGE 697] AND ADKINS AND GWATHMEY [PAGE 714])

DR. CHARLES W. LESTER, New York, N. Y.—If I seem to dwell on my points of disagreement with these papers it does not mean that I do not consider them excellent. The condition of funnel chest is so complex that conclusions cannot be reached from a limited group of cases, especially in a group of cases all before the age of puberty. Many funnel chests do not appear until the approach of adolescence and may not give symptoms before the age of 15. Yet operation on these patients produces such marked increase in exercise tolerance that they all recognize the improvement, functional studies to the contrary notwithstanding.

I believe that disproportionate overgrowth of the ribs is a factor in the late appearance of funnel chest, just as I believe that it is the most important factor in the protrusion deformities. Heredity is also a factor in both types of deformity and it is obvious that our concept of the etiology of anterior chest-wall deformities should be revised. That the pull of the diaphragm during inspiration is a dominant mechanism in producing funnel chest is obvious.

This diaphragmatic abnormality interferes with effective cough so that a minor respiratory infection can become major and pneumonitis or chronic bronchitis can result. If this occurs in infancy, operation should be undertaken regardless of the age and, in babies under one year of age, the simple sternoplasty should be used because it *is* simple. A radical sternoplasty may be necessary a couple of years later but, in the meanwhile, the baby has recovered from the infection and has had a chance to develop in a normal manner; and at least half of my patients who had simple sternoplasties have not required a radical sternoplasty later.

Furthermore, because the chronic pulmonary infection can produce a postoperative cough after radical sternoplasty which, with an unsupported sternum, can ruin a good result, I shall continue to pass wires under the sternum, carry them out onto the chest wall and fasten them into a "ladder" (Brown's terminology). This is for support and not traction. The support is easily applied, easily removed in a week or less and the procedure is less time-consuming than operations devised to avoid it. The danger of infection is highly overrated. I have never had an infection when wires were used and only once with a rib strut, and this did not compromise the result.

DR. J. KARL POPPE, Portland, Ore.—I rise to report the case of a 32-year-old woman with anginal pain on effort in whom the repair of a funnel chest deformity markedly decreased her symptoms and permitted her to recover from narcotic addiction. This operation was performed during the late 1940's before the substernal rib strut was being used. About 3 years later, the funnel chest deformity recurred, associated with recurrence of severe anginoid symptoms. These symptoms could be exaggerated by pressure over the depressed sternum.

This case serves to illustrate my contention that the surgical repair of a funnel chest deformity can produce improvement in cardiorespiratory disability in the adult.

DR. RALPH A. DORNER, Des Moines, Iowa.—I rise merely to put in a word for the use of a preserved rib as a strut rather than using a rib from the patient at the time of surgery. In a series of about 20 cases, I have used preserved ribs and feel that this can cut down on the operative time and operative trauma. Furthermore, the flat portion of an adult rib can be inserted beneath the sternum with considerably less difficulty than is encountered in using the young patient's rib with its much greater curvature. With the use of a rib strut, one can elevate the sternum and know that it will remain in place with an ultimate good result. The only complication in my series has been in one patient in whom I put the rib while it was still very cold. This patient developed a pleural effusion which was found to be sterile on aspiration and did not recur. I do not know whether the coldness had anything to do with the effusion or not, but I have been allowing the rib to warm as it is soaked in Terramycin solution prior to surgery.

I feel that with the use of a rib strut one can elevate the sternum and know that the condition will remain corrected. It is now 9 years since my first "strut" patient was operated upon.

DR. PAUL W. SANGER, Charlotte, N. C.—The pathophysiology in patients with deformed chests is hypoventilation, as it is with any type of thoracic cage deformity with a fixed chest wall, whether it be kyphosis, pectus excavatum (funnel chest), or pectus carinatum (pigeon breast). These individuals have lost the bellows effect of the chest which is absolutely necessary to bring a sufficient quantity of air for exposure to the gaseous exchanging surface of the lung. I think in the pectus excavatum and carinatum the deformity is created by overgrowth of the costal cartilages. The patients selected for operation are those with symptoms of breathlessness, palpitation, or with objective signs of paradoxical breathing, irrespective of age.

(slide) I do not think you should refuse to operate on a child just because he is young if he is having respiratory difficulty. Twenty-six of our patients have been under the age of 3 years.

(slide) Of the 103 patients we have operated upon without a single death, all were put through the gamut of physiologic studies, and we have found that a low pH was a consistent abnormality. Also, the pulmonary arterial pressure, even although it may be normal at rest, remains high for an abnormally long time following bicycle exercise of the legs.

I believe the cartilages should be removed entirely and only a small strip of the perichondrium, after it has been shortened, is attached to the undersurface of the sternum. I do not use any form of external fixation, nor do I object to entering the pleural cavity; as a matter of fact, I frequently drain the pleural cavity in order to avoid repeated pleural aspirations which are otherwise necessary.

This series represents patients we have operated upon in the last 13 years. Many of them have since married and some have even had service in the Marine Corps.

DR. MARK M. RAVITCH, Baltimore, Md.—I rise to emphasize what previous discussers have already stated, that this is a condition which causes symptoms, and that patients should be operated upon for relief of symptoms and prevention of the development of symptoms, as well as for correction of the cosmetic and postural deformity. We have seen adults, so severely symptomatic as to be in heart failure, relieved and returned to a symptom-free life after operation, and children, of all ages, with moderate degrees of limitation of ability for exercise.

(slide) This slide is of a child of 10 months of age who had a frighteningly severe congenital stridor which disappeared on the operating table and has not returned since operation. The stridor had been so severe that from moment to moment the necessity for tracheostomy had been entertained.

Up to the present, we have operated upon 73 patients for pectus excavatum. With respect to the ages of the patients operated upon, we feel that the operation should be undertaken in infants or children whenever the deformity is judged deep enough to merit operation, or whenever it has been observed to be progressive. In adults, we have operated only upon patients who are symptomatic. As you see from the slide, we have operated upon 9 children under the age of 12 months and a total of 23 under the age of 2 years. We have not observed any particular difficulty in operating upon these infants and find they tolerate the operation at least as well as the older patients. We have not required tracheostomy in any cases. We have operated upon 43 patients between the ages of 2 and 20, and upon 7 patients between the ages of 20 and 41. All the latter were symptomatic, several of them severely so, and all obtained complete relief following operation.

The cosmetic end result in children has always been satisfactory, although in some of the earlier cases, when we did not overcorrect them as much as we do now, the chest was not as full and round as we would like to see it. The cosmetic result in the adults has been less satisfactory and is limited by the fact that the manubrium itself is depressed, and that actually the clavicle seemed to be pointing backward and medially so that the forward corrective displacement of the body of the sternum is limited by the position of the manubrium.

With respect for the etiology of the deformity, we have assumed it to be due to overgrowth of costal cartilages. I show you this slide of a child with congenital absence of the abdominal musculature. This is essentially a preparation in which the pull of the diaphragm is unopposed by the rectus and may be considered comparable to an otherwrise exaggerated pull of the diaphragm. Here, at 3 months, you see no striking deformity of the sternum but, in the next slide, at 3 years of age, the distal end of the sternum is pointing sharply back where it has been pulled by the unopposed diaphragm. There is no sternal concavity. Furthermore, in many children with pectus excavatum the maximal depression is some distance cephalad to the insertion of the diaphragm.

From the standpoint of technique, we continue to exercise all of the deformed cartilages subperichondrially for the full extent of their deformity, to transect the first normal cartilage which is left behind, and to perform the sternal osteotomy at the interspace above this. This permits a three-point fixation: (1) across the sternal osteotomy, and (2) and (3) the resuturing of the transected cartilage. This produces a very firm fixation of the sternum and neither in infants nor in adults have we had to employ either external fixation or any form of strut.

I would suggest that perhaps the most important thing which has emerged from this morning's discussion is that today, in contrast with the situation at the time when Dr. Ochsner and Dr. DeBakey wrote their basic review, the situation is not whether these children should be operated upon, but when and how.

DR. LYMAN A. BREWER III, Los Angeles, Calif.—I have enjoyed the authors' presentation of these papers on pectus excavatum very much. We have been interested in this problem for a number of years and have carried out extensive cardiorespiratory tests including cardiac catheterization to determine the functional impairment. Our studies in a series of more than 60 patients reveal the fact that in the vast majority of cases, pectus excavatum is not a physiologically disabling condition. Although infants are difficult to test, there are very few infants and young children that show signs and symptoms of fatigue. Even in some of the teen-age patients and adults who have had moderate dyspnea, palpation, and fatigue on exertion, careful physiologic testing has not shown functional impairment of moment. Therefore, in our experience, the vast majority of operations that are being performed are carried out for psychological and cosmetic reasons. In the teen-age child, psychological and cosmetic reasons may be most important. Because teen-agers do indulge in sports and develop complexes out of all proportion to the physiologic impairment, surgical correction for the marked deformity is very definitely indicated. In those patients who have symptoms of cardiorespiratory deficiency, there is added reason for surgery.

We have employed an operative technique which makes all casts, external traction, temporary foreign bodies, splints, or fixation unnecessary. In this technique, we remove

perichondrally the deformed cartilages and subperiosteally the osseous portion of the deformed part of the sternum down to the posterior periosteum. A cardiolysis is carried out if there is displacement of the heart. The posterior periosteum of the sternum, which is an exceedingly tough, dense structure, is pulled upward by the muscle pull of the pectoralis major muscles by fixing the pectoralis fascia to the posterior periosteum of the sternum with a midline row of interrupted heavy cotton or silk sutures. No internal nor external splint or traction is thus needed. To increase the rapidity of sternal regeneration, bone chips of the resected sternum are placed in the periosteal bed. There is practically no paradoxical movement once the closure of the chest wall has been completed and the chest wall is completely flat with the deformity corrected as soon as the operation has been completed. The patient coughs and breathes well. The patient is up and about in a few days and may be discharged home in from 7 to 10 days following the operation. The postoperative period is usually very smooth, without serious complications or prolonged morbidity. The sternum is usually solid in from 6 to 8 weeks. It is important, of course, that the patient maintain good posture during the period of sternal regeneration, as it is for all types of sternoplasty. The shortened and simplified postoperative care without the necessity for long periods in casts with external traction or with external-internal splint, the excellent immediate cosmetic result, and the improved physiologic functioning of the thoracic cage commend the above technique for adults and children alike. We rarely operate upon infants and young children unless there is definite evidence of physiologic impairment, as pointed out by Dr. Ravitch.

DR. LAURENCE K. GROVES, Cleveland, Ohio.—The general practitioner and the pediatrician seem to be inclined to tell the parents of these children not to do anything about the deformity, it will probably go away. The only trouble with this advice is that it is not true; if anything, the deformity gets deeper with growth. We agree with most of the discussers of these papers this morning that frequently patients do have symptoms; however, it is usually difficult to demonstrate objective cardiopulmonary changes. In addition,, more often than not these people will not have otherwise well-formed chests and I think it is important to discuss with the parents the fact that we can cure the deformity, but that the child will still have a flat chest.

(slide) Here is a preoperative picture of a typical child we would operate upon.

(slide) We do essentially the same operation that has been described, but maintain external traction with rubber bands to a wire frame taped to the anterior chest. In children, this is necessary for only a week or two.

(slide) In the adult it is a little harder to attach the frame to the chest and it is incorporated in a light plaster body jacket.

(slide) Traction is maintained ordinarily from 4 to 6 weeks.

(slide) We have had the same experience in infants as cited by Dr. Ravitch. Here is a 12-week-old infant who had severe stridor. At rest, there was actually no deformity, but on inspiration there was tremendous retraction. Nothing was actually done to the pectus, but traction sutures were placed in the sternal cartilage and traction maintained for approximately 2 weeks. There was no further respiratory distress.

DR. RICHARD H. OVERHOLT, Boston, Mass.—Dr. Welch and the previous discussers have indicated that results in the repair of this deformity can be improved by, first, a careful selection of patients and, second, by simplification of the technique of the operation. There seems to be general agreement that older patients some form of temporary fixation or support to the elevated sternum is desirable. Our experience has been largely with adult or adolescent patients. Recently, I have provided internal fixation by inserting a Steinmann pin from one side of the cage to the other and beneath the sternum. Both ends of the pin are threaded to accept a metal washer which can be turned into proper position. They prevent lateral movement of the pin. The period of hospitalization averages under 2 weeks. The pin can be removed easily as an office procedure 2 to 3 months later. This method is simple, safe, and effective.

DR. JAMES E. DAILEY, Houston, Texas.—I will take only a moment. Our experience with the strut in the majority of cases has been with children between the ages of 4 and 7. I would like to show you the youngest one who was 30 months old.

(slide) This is the child 10 days postoperatively.

(slide) This slide illustrates the rib strut wired in place.

(slide) The catheter you see in at the upper right leads to the space beneath the sternum and is placed there for about 24 to 36 hours to get rid of the fluid and dead space beneath the sternum.

DR. WELCH (Closing).—I would like to thank the discussers for their interest and the helpful information added to the subject of "pectus excavatum deformity." There are just one or two closing comments that I would like to make.

The problem of recurrence has perplexed us. We have encountered this complication five times. It does not occur in the immediate postoperative period when external traction devices are used. It does not even occur in the first month when internal stabilization with a Kirschner wire is used. All patients appear satisfactory up to 3 months. When you see them 6 months to a year after operation, there, unexpectedly, is the patient with recurrence.

The second comment has to do with the type of operation. I have a very healthy respect for the risk associated with surgical correction of this disease, especially in the younger group. I believe that the more extensive operations involving a complete takedown of the anterior chest wall, with violation of both pleural cavities, will set the stage for major pleural cavity and mediastinal complications. I doubt that 50 consecutive operations of this type can be carried out without a mortality. Consequently, we feel that a simple external operation, of the type illustrated today, is preferred. A satisfactory thoracoplasty result is attained with virtually no postoperative complications.

The third comment has to do with late troubles. This has been adequately documented in the second and third decades of life. They do not develop clinically and cannot be objectively identified in the first decade. For this and for the other reasons presented, we recommend that operation be undertaken in selected individuals at or near the age of 3.

DR. GWATHMEY (Closing).—On behalf of Dr. Adkins and myself, I would like to thank the discussers. We agree with Dr. Dorner that the operative time is shortened by the use of preserved ribs in place of fresh bone. Recently, we have been using struts made of stainless steel and notched at both ends for fixation in the experimental animal. At the present time, tissue reaction has been minimal and there has been no problem with infection. We hope to report on this at a later date.

HYPOVENTILATION, HYPOXIA, AND ACIDOSIS OCCURRING IN THE ACUTE POSTOPERATIVE PERIOD

R. Maurice Hood, Commander (MC) USN (by invitation), and
Arthur C. Beall, Jr., Lieutenant (MC) USNR (by invitation),
Oakland, Calif.
(Sponsored by Frank A. Gerbode, M.D., San Francisco, Calif.)

ADEQUATE VENTILATION during thoracic operations has long been the goal of surgeons and anesthesiologists. Crafoord seems to have been among the first to realize the importance of adequate ventilation as opposed to that required to maintain life during open-chest operations. Acidosis during anesthetic administration and surgical procedures is not a newly recognized phenomenon. Austin and co-workers,[1] in 1922, Cullen and associates,[2] in 1924, Stehle and Bourne,[3] in 1924, and Leigh,[4] in 1942, have all observed acidosis and a rise in pCO_2 following the administration of general anesthetic agents. However, the importance of acidosis during surgical procedures and its frequency and severity do not seem to have been appreciated until the work of Beecher[5] was reported before this group in 1949. Subsequently, numerous studies have been done, the most notable among which have been the work of Gibbon and co-workers,[6] Ellison, Ellison, and Hamilton,[7] Miller and associates,[8] Stead, Martin, and Jensen,[9] and Taylor and Roos.[10]

These investigators have clearly shown that hypoventilation is responsible for CO_2 retention with the development of respiratory acidosis. Attempting to prevent this phenomenon, much work has resulted in methods and efficiency of ventilation during intrathoracic procedures far superior to those available 15 years ago.

Little investigation has been directed toward respiratory efficiency during the immediate postoperative period. In Gibbon's study[6] (1950), 11 patients had blood samples drawn 1 to 2 hours postoperatively; of the 11 cases, 2 showed a decrease in pH after surgery and, in 1 patient, O_2 saturation dropped to 78 per cent.

Martin and Stead,[11] in 1953, removed blood samples in 18 patients 20 to 30 minutes after the patient had been returned to the ward. All but 1 of these

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showed a pH drop from the preoperative level but since no samples were taken at the end of surgery, the respiratory efficiency of the postoperative period alone could not be evaluated. However, their mean values of CO_2 excretion and alveolar ventilation do not indicate hypoventilation in the group as a whole.

Sealy, Young, and Hickham[12] reported 2 patients, both of whom had respiratory acidosis in the postoperative period, possibly aggravated by the administration of oxygen. Respiratory assistance with other measures were used to relieve these patients.

Hamilton and Devine[13] determined pCO_2 from alveolar air and measured minute volumes in 100 surgical patients. No reference is made concerning the number of thoracic surgical patients included. Their studies indicated that 25 per cent of the total number had pCO_2 levels in excess of 45 mm. Twelve of 25 patients, with increased pCO_2 values, were shown to be hypoventilating in the recovery room.

We have been impressed with the apparent observation that thoracic surgical patients in the recovery room were hypoventilating; therefore, this study was undertaken to determine accurately the characteristics of ventilation during the first postoperative hour and to determine if hypoventilation was present in a sufficient degree to alter O_2 saturation, pCO_2 values, and pH of arterial blood.

METHOD

Thirty-eight patients were studied. Thirty underwent intrathoracic operations of miscellaneous types (Table I). Eight were individuals having major abdominal procedures. The subjects were not studied consecutively and no patient known or thought to have measurable respiratory insufficiency preoperatively was included.

TABLE I. TYPES OF OPERATIONS PERFORMED

TYPE OF OPERATION	NUMBER
Thoracic Surgical Patients	
Pneumonectomy	2
Lobectomy	5
Segmentectomy	6
Bronchoplasty	1
Mitral commissurotomy	4
Closure, atrial septal defect	1
Resection, mediastinal tumor	2
Vagotomy	1
Cervicodorsal sympathectomy	2
Resection of chest wall tumor	3
Pleurectomy	2
Exploratory thoracotomy	1

An arterial blood sample was obtained on the day prior to operation, and at the end of the procedure an intra-arterial needle was inserted into a femoral artery. Samples were obtained just prior to the removal of the intratracheal tube and at 15-minute intervals for 1 hour. Samples were analyzed for O_2 content and capacity after the method of Van Slyke and Stadie[14]; CO_2 content was determined following the method of Van Slyke and Neill.[15] These were

converted to serum content values using the system of Van Slyke, Sendroy, and Liu.[16] Serum CO_2 tension in millimeters of mercury was calculated, using the normogram of Van Slyke and Sendroy.[17] Heparinized blood samples were collected in pipettes containing sodium fluoride, as suggested by Graig and coworkers,[18] and the pH was determined directly, using a Cambridge glass electrode pH meter. In addition, spirograms of 2-minute duration were made on each patient, to coincide with each of the 4 blood samples drawn in the postanesthetic period. From these, tidal volume and minute ventilation volumes were measured. Alveolar ventilation was estimated using the values for anatomic dead space determined by Fowler.[19]

Anesthesia in this group of patients was conducted by 2 anesthesiologists and 3 residents in anesthesiology, who were uninformed as to the purpose or results of the study. Pre-anesthetic medication usually consisted of Demerol, pentobarbital, and atropine; occasionally morphine and scopolamine were used.

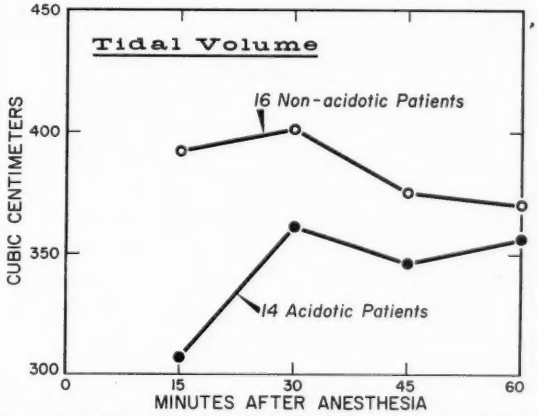

Fig. 1.—Comparison of the relationship between the mean values for tidal volume in 14 thoracic surgical patients who acquired acidosis in the postoperative period with 16 who did not show this phenomenon.

Anesthetic agents employed were usually intravenous Pentothal Sodium for induction and nitrous oxide and oxygen inhalation, supplemented by intravenous succinylcholine hydrochloride for the duration of the procedure. Cyclopropane and ether were used in a few patients. Controlled respiration was used in all thoracic cases; in 16 patients this was carried out by manual compression of the anesthetic bag and, in 14 patients, controlled ventilation was administered by the Jefferson ventilator with intermittent positive-negative pressure.

RESULTS

Table II gives the data acquired on the total series of patients. From this material, 14 patients in the thoracic series were identified as demonstrating a drop in pH of at least 0.10 unit during the first postoperative hour. Several of the other 16 showed lesser changes, no change, or a rise from a lowered pH

TABLE II. DATA ON 38 PATIENTS STUDIED

CASE NO.	NAME	SEX	AGE	ARTERIAL O_2 (% SATURATION) PREOPERATIVE CONTROL	END OF ANESTHESIA	INTERVALS (MIN.) 15	30	45	60	ARTERIAL pCO_2 LEVELS (MM. HG) PREOPERATIVE CONTROL	END OF ANESTHESIA	INTERVALS (MIN.) 15	30	45	60
Thoracic Surgery															
1	L. S.	M	30	90	100	100	95	94	91	35	42	43	42	--	36
2	N. J.	M	27	98	100	89	92	93	93	33	27	40	46	52	48
3	O. O.	M	57	87	100	97	91	--	86	35	38	43	42	--	36
4	E. C.	M	39	90	90	90	92	91	88	38	44	48	47	49	49
5	G. D.	F	39	92	100	100	82	93	88	29	32	58	49	44	44
6	M. E.	F	14	91	100	92	94	96	91	29	38	45	42	52	40
7	G. H.	M	62	91	100	89	82	87	78	30	35	46	34	38	41
8	P. P.	M	42	91	100	76	91	90	89	35	28	54	52	56	53
9	I. B.	M	33	96	100	87	86	88	86	46	33	46	48	47	45
10	W. S.	M	35	92	100	88	86	87	84	45	32	42	50	49	46
11	E. E.	F	31	94	95	100	92	94	95	36	45	39	52	44	47
12	D. M.	M	43	94	100	88	89	89	87	31	33	42	--	--	46
13	R. H.	M	37	91	100	98	91	87	89	42	51	46	45	42	43
14	E. L.	M	54	86	99	97	95	95	90	37	34	53	40	--	40
15	R. H.	M	35	91	100	97	97	100	98	34	39	46	43	45	44
16	D. M.	M	22	88	100	94	96	97	93	52	34	48	48	46	47
17	T. D.	M	20	91	100	100	100	97	98	37	51	45	47	44	44
18	A. B.	M	19	93	100	100	100	97	85	52	36	38	37	36	35
19	O. B.	M	39	89	95	85	87	91	89	35	59	41	46	--	41
20	E. R.	M	39	88	100	88	91	91	89	32	40	40	45	37	37
21	G. M.	F	38	93	100	91	89	--	86	32	37	30	42	--	28
22	E. W.	F	26	96	100	98	91	96	89	32	39	48	42	40	38
23	W. D.	M	32	95	100	--	100	98	99	34	42	--	40	44	44
24	J. R.	M	19	92	71	78	78	74	80	34	86	58	58	46	49
25	R. E.	F	26	93	92	84	86	86	76	32	43	74	80	67	62
26	V. H.	F	35	98	100	96	98	95	94	41	31	32	35	33	29
27	R. M.	M	49	--	100	88	83	90	99	--	36	33	33	22	48
28	J. S.	F	22	90	100	98	100	97	95	40	37	46	46	47	43
29	W. C.	M	33	93	100	93	95	92	95	--	53	49	44	39	41
30	L. H.	M	26	94	100	91	82	94	91	42	35	44	50	41	44
Abdominal Surgery															
31	M. K.	F	31	90	100	59	92	--	--	37	45	36	48	--	--
32	M. S.	M	59	87	100	100	97	88	85	36	51	49	45	38	46
33	A. S.	F	46	89	100	95	98	96	94	32	32	32	36	35	33
34	C. N.	M	36	91	100	99	94	90	91	41	46	49	51	44	44
35	C. M.	M	22	94	100	98	100	93	93	45	42	43	48	31	54
36	J. S.	M	30	94	100	100	100	95	95	27	50	35	42	37	--
37	D. R.	M	52	91	100	95	93	96	98	39	28	33	28	29	29
38	L. K.	M	26	91	100	98	94	97	97	35	41	35	34	35	33

*Jefferson.
†Recovery room.

acquired during the period of anesthesia. The spirograms and ventillatory values of these groups of patients were studied individually and mean values for the group calculated. Fig. 1 compares the mean tidal volumes of the 2 groups at 15-minute intervals and clearly indicates significantly lower values for the patients in the acidotic group. Some individual values were measured in which the tidal volume was as low as 150 c.c.

Fig. 2 is a similar comparison of the mean values for minute ventilation of the same 2 groups. These values show an even greater divergence and, in addition, illustrate the usual trend toward normal values for patients in the depressed group as the time after anesthesia progressed.

Alveolar ventilation is without doubt the most important measurement

During the Immediate Postoperative Period

ARTERIAL pH VALUES								
PREOPERATIVE CONTROL	END OF ANESTHESIA	INTERVALS (MIN.) 15	30	45	60	VENTILATOR* USED	NASAL O_2 GIVEN IN R.R.†	PROCEDURE
7.43	7.31	7.29	7.32	7.35	7.34	yes	yes	Pleurectomy
7.35	7.37	7.32	7.28	7.22	7.26	yes	no	Sympathectomy, upper dorsal
7.42	7.40	7.28	7.28	--	7.37	no	yes	Wedge resection, LUL
7.42	7.35	7.33	7.31	7.30	7.30	no	yes	Bronchoplasty, left main
7.48	7.43	7.22	7.28	7.32	7.32	no	yes	Mitral commissurotomy
7.47	7.43	7.33	7.36	7.28	7.37	no	yes	Segmentectomy, RUL
7.51	7.40	7.32	7.43	7.40	7.36	yes	yes	Lobectomy, LLL
7.36	7.43	7.23	7.24	7.20	7.21	no	no	Transthoracic vagotomy
7.34	7.42	7.31	7.28	7.28	7.28	no	no	Wedge resection, RUL
7.39	7.44	7.32	7.28	7.29	7.31	no	yes	Closure, atrial septal defect
7.46	7.29	7.31	7.24	7.30	7.29	no	no	Resection mediastinal tumor
7.43	7.33	7.29	--	--	7.23	yes	no	Segmentectomy, LUL
7.40	7.29	7.33	7.35	7.37	7.36	no	no	Lobectomy, LUL
7.43	7.48	7.26	7.40	--	7.39	yes	yes	Pneumonectomy, right
7.47	7.38	7.30	7.34	7.34	7.31	yes	no	Resection mediastinal tumor
7.29	7.46	7.33	7.34	7.36	7.35	yes	yes	Lobectomy, RU & ML
7.44	7.32	7.37	7.35	7.37	7.37	no	yes	Excision chest wall tumor
7.31	7.43	7.40	7.41	7.41	7.42	yes	yes	Segmentectomy, LUL
7.43	7.31	7.39	7.32	--	7.38	no	yes	Sympathectomy, cervicodorsal
7.53	7.38	7.35	7.32	7.42	7.41	yes	yes	Pleurectomy
7.40	7.32	7.41	7.26	--	7.44	no	yes	Mitral commissurotomy
7.47	7.29	7.20	7.27	7.31	7.33	yes	yes	Segmentectomy, RLL
7.49	7.37	7.32	7.39	7.34	7.34	no	yes	Segmentectomy, LLL
7.50	7.13	7.28	7.27	7.36	7.33	no	yes	Lobectomy, LLL
7.48	7.20	7.02	7.02	7.09	7.12	no	yes	Lobectomy, ML
7.36	7.46	7.40	7.38	7.40	7.48	no	yes	Mitral commissurotomy
--	7.46	7.45	7.41	7.52	7.29	no	yes	Exploratory thoracotomy
7.37	7.39	7.31	7.31	7.30	7.34	yes	yes	Pneumonectomy, left
--	7.23	7.26	7.32	7.32	7.33	yes	yes	Mitral commissurotomy
7.38	7.39	7.35	7.32	7.36	7.34	yes	no	Resection chest wall tumor
7.37	7.26	7.42	7.23	7.23	--	no	part time	Cholecystectomy
7.40	7.35	7.30	7.34	7.40	7.28	no	yes	Cholecystectomy
7.49	7.41	7.43	7.38	7.40	7.41	no	no	Colectomy, right
7.37	7.30	7.28	7.25	7.30	7.32	no	yes	Gastrectomy, subtotal
7.34	7.37	7.34	7.27	7.52	7.26	no	yes	Exploratory laparotomy
7.53	7.24	7.37	7.32	7.39	--	no	yes	Cholecystectomy
7.32	7.51	7.42	7.48	7.48	7.48	no	no	Abdominoperineal resection
7.39	7.33	7.36	7.38	7.36	7.39	no	yes	Gastrectomy, subtotal

when evaluating adequacy of ventilation. The methods of determination vary, depending upon the method used in estimating dead space. For a study such as this to have practical value, the more elaborate methods of estimating physiologic dead space would have to be discarded. As previously stated, the writers have chosen to use the normal values for anatomic dead space recorded by Fowler.[19] The values so obtained were included in the formula; tidal volume minus dead space times rate equals alveolar ventilation. At low tidal volume values, Briscoe, Forster, and Comroe[20] have shown that this formula is not strictly applicable because the moving column of air in the tracheobronchial tree moves as a conical front and therefore some alveolar ventilation occurs even when tidal volumes are less than the calculated dead space. However, the estimated values so determined are more indicative of the true status of the

patient's ventilation than either tidal volume or minute ventilation values. Fig. 3 illustrates the mean alveolar ventilation values in the 2 groups previously compared. Again there is a significant difference in the two series.

The spirograms of individual patients graphically demonstrate the inadequacy of ventilation better than the values just discussed. There is considerable

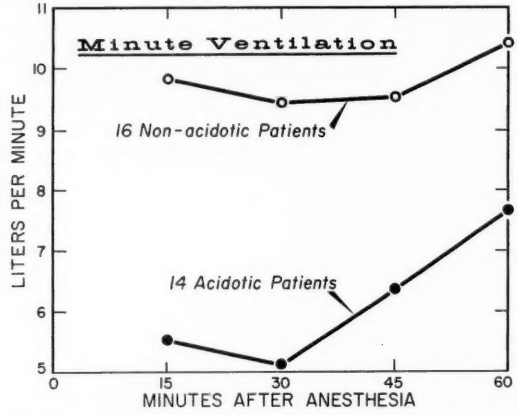

Fig. 2.—Comparison of the relationship between the mean values for minute ventilation in 14 thoracic surgical patients who acquired acidosis in the postoperative period with 16 who did not show this phenomenon. In addition to the obvious depression of the acidotic patients, the trend toward improvement during the hour of observation should be noted.

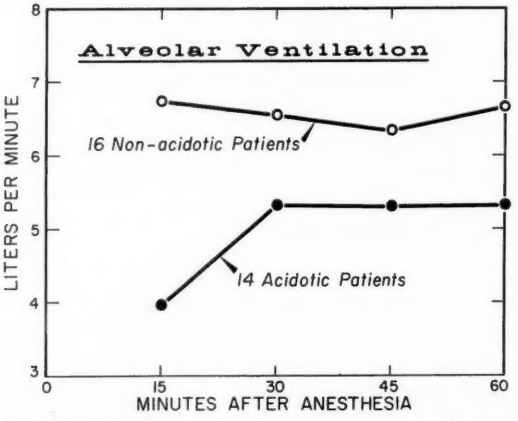

Fig. 3.—Comparison of the mean values from the estimation of alveolar ventilation in the 2 groups of patients compared in Figs. 1 and 2, and again showing an obvious difference, particularly in the first 15-minute period.

individual variation, as will be noted in Figs. 4 and 5. Inadequate ventilation resulted from low tidal volume, decreased rate, or from gross irregularity with periods of apnea as long as 25 to 30 seconds.

Hypoxia, although present in minimal degrees in several patients, was

largely prevented by the use of O_2 by nasal tube in the recovery room. However, O_2 saturation dropped to levels as low as 75 per cent in 2 patients who were receiving O_2; in these, the tidal values were at ranges from 150 to 255 c.c.

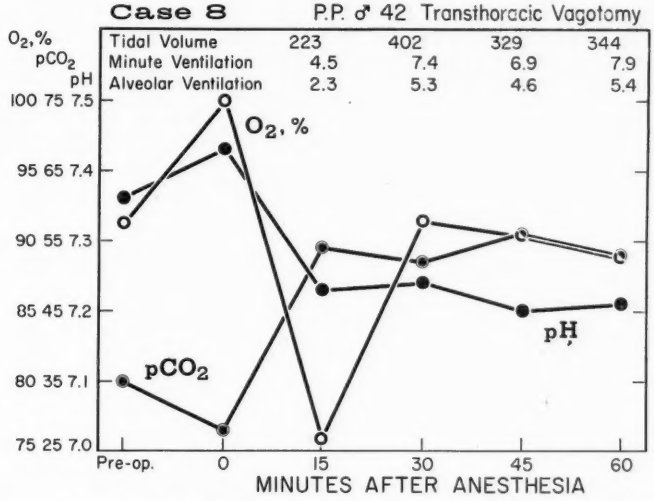

Fig. 4.—The total values obtained in Case 8, showing the development of respiratory acidosis and hypoxia during the recovery room period.

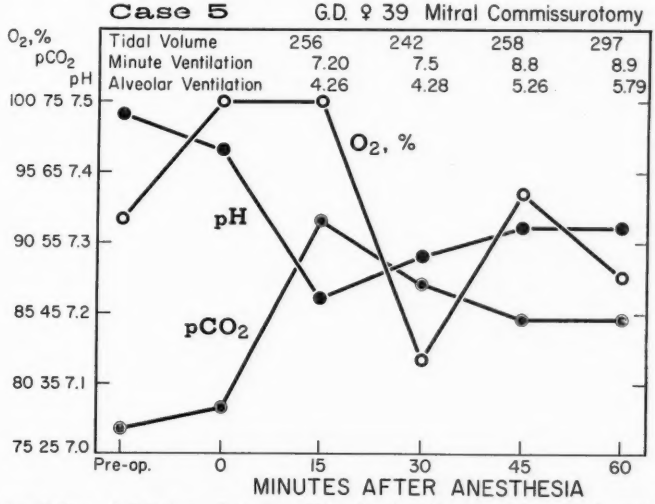

Fig. 5.—Values obtained in Case 5, again showing the development of respiratory acidosis and hypoxia coincident with low tidal volumes.

Evaluation of the effect of the use of the Jefferson ventilator indicates that of 14 patients in whom it was used, 7 fell into the acidotic group and 7 in the nonacidotic group. These figures indicate that the method of ventilation during

surgery had no effect on postoperative respiration. An incidental observation was that no patient in whom the Jefferson ventilator was used had marked acidosis at the end of surgery whereas there were 2 patients with pH levels of

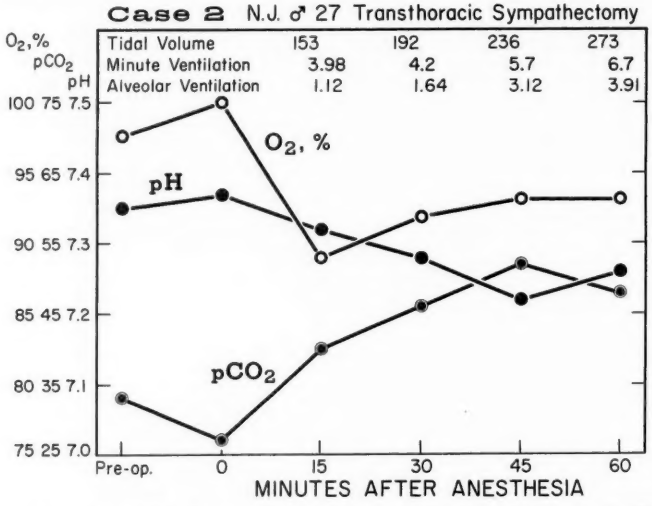

Fig. 6.—The values obtained in Case 2, demonstrating increased levels of pCO_2 and lowering of pH over a much longer period, in comparison with the 2 cases in Figs. 4 and 5.

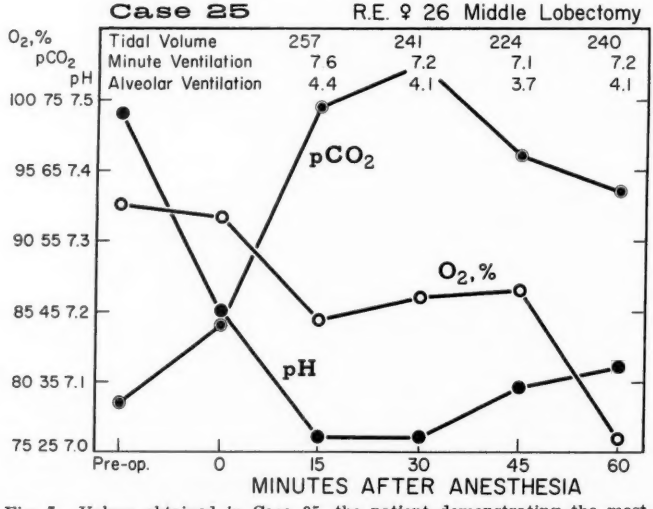

Fig. 7.—Values obtained in Case 25, the patient demonstrating the most marked deviation from normal values of any patient in the entire group. She acquired acidosis during the operative period; however, during the postanesthetic period, the pCO_2 levels arose to 80 mm. Hg and the pH dropped to 7.02.

7.20 or lower, who were ventilated manually. There appeared to be no correlation between the duration of the period of anesthesia and the incidence of respiratory depression.

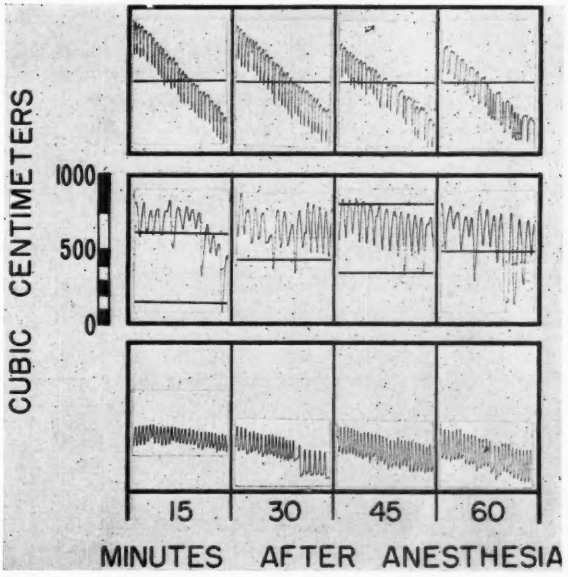

Fig. 8.—Sample spirograms from 3 patients in the acidotic group, showing generally low tidal volumes and gross irregularity in volume from breath to breath (note Case 30). In Case 2, a more commonly occurring situation may be seen—shallow, rapid, regular respiration in which the tidal volume gradually increased during the hour of observation.

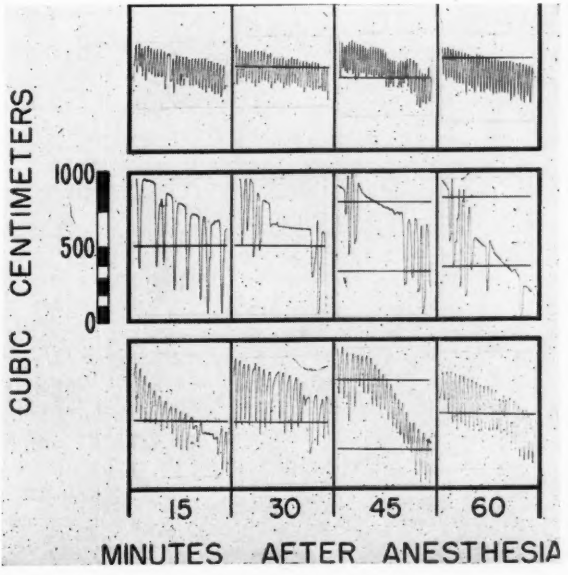

Fig. 9.—Sample spirograms of 3 other patients in the acidotic group, shown to demonstrate the long periods of apnea occurring in Case 35.

Figs. 4, 5, 6, and 7 are graphic demonstrations of the total values in 5 patients who were in the acidotic group.

Figs. 8, 9, and 10 are reproductions of the spirograms produced by 9 patients who became acidotic during the recovery room period.

DISCUSSION

Obviously the data obtained in this study are referable only to patients given one method of anesthesia by one group of anesthesiologists and therefore conclusions reached do not necessarily apply to all thoracic surgical patients. However, it would seem reasonable to assume that similar situations frequently prevail, and particularly so in patients with disease states impairing pulmonary function.

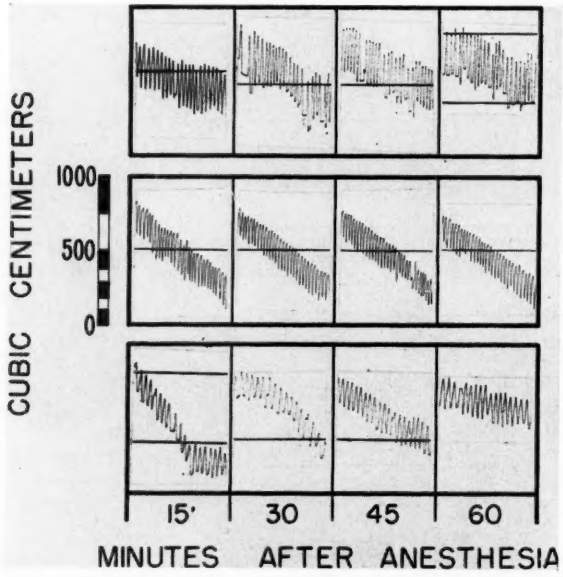

Fig. 10.—Sample spirograms of 3 other patients who became acidotic during the period of observation and showing the low tidal volumes, particularly in Cases 25 and 28.

The causes of respiratory depression in the immediate postanesthetic period are not easily separated or identified. It is possible that preanesthetic medication still may be effective during this period. The anesthetic agents certainly do not terminate their effect immediately upon cessation of their administration. In the series of Hamilton and Devine,[13] the greatest number of patients who were hypoventilating were those receiving nitrous oxide, Pentothal, and relaxants. A majority of the patients in this series were similarly anesthetized. However, this is not considered sufficient evidence to incriminate the agents used. Postoperative pain may be and probably is a factor; however, it was noted in this group of patients that as the hour of observation passed and pain apparently

increased, ventilation improved; also, that narcotics administered to relieve pain did not improve ventilation and occasionally appeared to impair it further.

It is noted that respiratory depression in this series was chiefly confined to those having chest operations. One obvious difference in the two groups, other than the surgical procedure, was the greater use of controlled respiration in those having thoracic procedures while the abdominal surgical patients were not rendered apneic for as prolonged periods as those with chest operations. All anesthesiologists are familiar with the fact that patients who have been in an apneic state resume normal respiration gradually and occasionally quite slowly. Whether or not this mechanism was still acting in the first operative hour was not determinable.

Whatever the cause or causes of respiratory depression, the resultant CO_2 retention and acidosis are considered of serious import even though they might occur only occasionally. Hypoventilation in patients with preoperative respiratory insufficiency, such as might be the case with emphysema or any other advanced parenchymal disease, might seriously affect the patient's chances of survival.

Certainly, sufficient experimental and clinical investigations have shown the lethal hazard of hypercapnea, particularly when associated with even a brief period of hypoxia. The fact that no patient in this series suffered any apparent ill effect from the abnormal acid-base balance indicates again the often demonstrated ability of the human organism to withstand physiologic trauma.

The practical application of this study should be an attempt at prevention or correction of hypoventilation. To anesthesiologists the idea of having the patient recover from the effect of the anesthetic agents as soon as possible after the termination of surgery is not a new one. However, despite the fact that, by the end of the first 15-minute period, every patient in this series had recovered sufficiently to obey simple commands and reject a mouth airway, the respiratory function was still impaired. Referring again to Table II, it will be noted that the majority of the hypoventilatory patients were in the first half of the series. As the anesthesiology department gradually became aware of the purpose of the study, more strenuous efforts to effect more complete and rapid recovery were made. Whether or not stimulating drugs are indicated for this purpose is controversial.

It is well known that hypoxia during hypoventilation can be largely prevented by the use of oxygen by almost any method. The results of this investigation bear this out. Despite the general recommendations for the use of oxygen during anesthetic recovery, the recovery room personnel were frequently found to be careless in its use, particularly if the patient was awake, not cyanotic, and in no apparent distress. No patient in the entire group gave any clinical indication of the abnormal physiologic state. There were no episodes of hypotension, no arrhythmias, and no cyanosis except in one individual who vomited and aspirated mucus. Therefore, the diagnosis of respiratory acidosis and hypoxia is not clinically feasible unless special apparatus for measuring alveolar CO_2 and O_2 saturation are available. However, hypoventilation can be detected

once the recovery room personnel are cognizant of respiration in terms other than rate and cyanosis. Numerous devices are available which are capable of measuring tidal volume, one of which (a basal metabolism machine) is available in most hospitals. The nursing staff can be easily taught to measure tidal volumes and to realize that values under 350 c.c. or rates under 15 per minute require notification of the anesthesiologist or surgeon.

Correction of hypoventilation may be accomplished most easily by assisted respiration which again may be administered by several means. Assistance with the bag of the anesthesia machine or the use of intermittent positive pressure valves such as the Bennett are most practical.

SUMMARY

1. The respiration of 38 patients—30 having had thoracic operations and 8 having had major abdominal operations—has been studied by spirograms made at 15-minute intervals for 1 hour after discontinuance of anesthesia. Arterial blood samples were taken at the same intervals and the O_2 saturation, pCO_2 levels, and pH values determined.

2. Fourteen of the 30 patients undergoing thoracic surgery were found to be hypoventilating to a degree sufficient to lower pH by at least 0.10. The pCO_2 values were correspondingly elevated.

3. Minimal to moderate degrees of hypoxia were also noted. Spirograms demonstrate hypoventilation and breathing irregularity. Possible mechanisms of respiratory depression are discussed. Emphasis on detection of hypoventilation and its correction by assisted respiration is suggested.

The authors gratefully acknowledge the technical assistance of Mrs. M. E. Hutchin, Chief Biochemist, and other members of the research laboratory and, also, the editorial assistance of Mrs. M. F. Jack, Clinical Investigation Center.

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DISCUSSION

DR. CLARENCE CRAFOORD, Stockholm, Sweden.—I am very thankful to have the privilege of the floor. Last year, as some of you know, I was not in a condition to come here to attend the meeting and I must say how glad I am to have the opportunity to attend this year.

I think the question that has been raised is important, especially in the group of open cardiac surgery cases. Postoperative hypoventilation is common in these patients. Therefore, we follow them very carefully in the postoperative period to control their respiratory activity.

In the small group of open-heart cases that we have had—it is now about 60—there has been a number of patients in whom we have found that ventilation postoperatively was not adequate, and that they run into CO_2 accumulation soon after operation. We follow them with repeated tests continuously during the first postoperative days. We believe that tracheostomy is valuable in all patients who do not recover their normal respiration spontaneously. We use an airtight fitting tracheostomy tube and connect the patient to an Engström respirator which takes over the patient's breathing, and keep them on artificial respiration, as is done in the paralytic cases of infantile paralysis, for a considerable time until we can measure that the respiratory exchange is normal. Sometimes that takes only a few hours; the longest time was about a week before the patient could be taken out of the respirator and be allowed to breathe by himself.

We are quite convinced that many cases that now end fatally can be helped through the postoperative period by these means.

DR. FRANK COLE SPENCER, Baltimore, Md.—I would like to describe briefly some experiences with acidosis in surgical patients. We have made several hundred pH determinations during the past 2 years as such studies are routinely done for several hours following operation under hypothermia or cardiopulmonary bypass. The most impressive feature has been failure of clinical findings to suggest acidosis. The patients whose pH is 7.20 to 7.10 are semiconscious; lower pH values (7.10 to 7.0) are often associated with coma, and even respiratory arrest. The most dangerous aspect of this situation is that, without much warning, respiratory arrest may suddenly occur in some patients. Because of the absence of clinical signs of acidosis, and because of the danger of respiratory arrest in such patients, we think it is useful to measure the pH in all patients who are comatose, dyspneic, or hypotensive from unexplained causes. Severe acidosis has been found in patients with hemothorax with mediastinal displacement, patients with respiratory insufficiency, or patients with

severe hypotension from hemorrhagic shock. The acidosis can be corrected with increased ventilation or intravenous sodium bicarbonate, 3 to 8 Gm., after which the underlying cause of the acidosis can be treated.

Like Dr. Crafoord, we have also come to increasing use of the respirator. It is particularly useful in patients with severe cardiac failure, and we have had some of these patients in a respirator for 3 to 4 weeks until they made a complete recovery.

DR. GEORGE H. A. CLOWES, Jr., Cleveland, Ohio.—I rise simply to emphasize the dangers of the acidosis described by Drs. Hood, Beal, and Gerbode. Uncompensated acidosis is a lethal affair if carried beyond a certain point. It renders the organism incapable of responding to superimposed stresses in the normal fashion. A series of experiments performed in our laboratory last winter by Miss Mary A. Hughes, a medical student, will serve to illustrate this. Acidosis was induced in dogs by the infusion of hydrochloric acid intravenously. Little or nothing occurred to the circulation until the arterial pH fell below 6.9 when heart failure usually took place. However, at arterial pH values ranging from 6.9 to 7.1, it was found that animals were quite incapable of responding to blood loss. They entered a state resembling "irreversible" or normovolemic shock from which they could only be recovered by the infusion of sodium bicarbonate.

(slide) Dr. Hood has demonstrated acidosis to be an immediate postoperative affair. The data from this patient, who happens to have had a circulatory bypass and cardiotomy, will serve to illustrate the course of events following any major thoracotomy. During perfusion there was some accumulation of lactic acid with a fall in CO_2. Two hours postoperatively the lactic acid had fallen and CO_2 was up slightly. Note that the following morning lactic acid was considerably elevated, probably due to the arterial oxygen desaturation (85 per cent) and a moderate impairment of cardiac function. Yet CO_2 was low and the arterial pH normal.

In our experience this is common. But if one finds an uncompensated acidosis with an accumulation of CO_2 as well as lactic acid, death will supervene shortly unless strenuous measures are taken to improve the respiration and the acid-base balance.

DR. RALPH ADAMS, Wolfeboro, N. H.—I wish to testify to the enormous practical importance of this presentation by Dr. Hood. It is applicable to all cases, not only as a method of study, but also as a guide to therapy. First, consider those cases which are complex; no one needs to have his attention directed to acidosis or hypoxia as a possibility in the complex case, such as a patient coming off the pump-oxygenator. But it is well to remember simpler situations, such as that of a patient who has a perforated ulcer closed surgically, and who is back in the ward 45 minutes after receiving his preanesthetic medication, because things went better than anticipated. His medication strikes him after he gets back and he does not breathe. Or the case of a patient being prepared for bronchoscopy, who has overreacted to the highly effective combination of narcotic, sedative, and tranquilizer one is currently using, and then he does not breathe. These are examples of simple cases; however, death ensues just as surely as it does in complex ones.

Second, one can recognize them as being close to trouble in a less complex way than by pH figures, fortunately. These are helpful and we like them, but one can watch the patient's breathing; it does not take a highly educated person to do it. We have found that ward aides are very good at telling whether patients are breathing or not. People generally are poor at recognizing cyanosis; if even suspected, our rule is to assume it to be present and to treat for it. Oximeters and Van Slyke analyzers will show scientifically what must be treated at onset, or even better, on suspicion.

Third, there are several ways by which one can assist the patient therapeutically. One need not be as scientific as is required in conducting excellent laboratory studies such as those we have heard about in this paper. Even the ward aide can help oftentimes those patients who are slightly acidotic, who are semiconscious, but who generally can be persuaded to breathe. If they cannot be persuaded to breathe, they require assistance. One remembers artificial respiration, and the numerous good ways of applying it. A little chest wall assistance

for a few minutes often is all that is needed. Then there is the demand valve; nurses can be taught to use this equipment as efficiently as the most highly trained physician. The fact that there is no nurse or ward aide on my surgical wards who cannot use the demand valve, as well as the chief surgeon, I think has saved a number of lives. Demand valves are not very expensive and are available in differing types from several manufacturers. Suction can be applied intermittently to the thoracic wall and it moves the wall harmlessly when there is the long need for assistance that is required by some postoperative patients before they breathe spontaneously in adequate volume. The Emerson combination of a plastic raincoat and vacuum cleaner is one extremely simple and almost hazard-free way of giving such assistance.

DR. HOOD (Closing).—I would like to thank the discussers for their comments. We certainly agree with Dr. Clowes that this is not a short-term problem. The majority of our patients studied who were abnormal had little or no improvement in the first postoperative hour. This observation has been made before, particularly by Björk, who studied the respiratory deficiency one to 2 days following various surgical procedures and found the oxygen saturation still lowered after this time. We would also like to emphasize that only in the larger hospitals and medical centers are there facilities for analyzing CO_2 or determining pH. We would agree with Dr. Adams that detection of hypoventilation is a practical problem which even relatively inexperienced personnel can be taught to accomplish. Numerous devices are available which can be used to measure tidal volume; one of these, a basal metabolism machine, is present in most hospitals and can be used by nurses, so this problem can certainly be solved without resorting to more complicated apparatus.

I would like to thank the Association for the opportunity of presenting this paper, and particularly Dr. Gerbode for his personal interest and encouragement.

CHONDROSARCOMA IN THE POSTERIOR MEDIASTINUM

John J. Sullivan, Jr., M.D., and Joseph L. Mangiardi, M.D.
Bethpage, N. Y.

CARTILAGINOUS neoplasms of the thorax are relatively common, nearly always grossly involve osseous structures (sternum, vertebra, or rib), and are most often malignant. O'Neal and Ackerman,[1] in a review of the clinical and pathologic aspects of these lesions, presented 96 cases including two chondrosarcomas which occurred in the posterior mediastinum causing rib destruction. In 1950, Weisel and Ross[2] described excision of a malignant cartilaginous tumor with hourglass involvement of the mediastinum and spinal canal. There was gross destruction of vertebral bodies, pedicles, and laminae, as well as rib heads. In 1956, Ringertz and Lidholm[3] reported one chondrosarcoma from a personal series of 155 mediastinal neoplasms and cysts. This was posterior in location, probably originating from a vertebral body, and recurred 2 years after excision. In 1956, Aronstam and his associates[4] described excision of a chondrosarcoma from the posterior mediastinum in 2 cases in which origin from a vertebral body was demonstrated. In each of the 6 foregoing cases, including those of O'Neal and Ackerman, gross destruction of cortical bone was demonstrated by x-ray studies or at operation.

The occurrence of a circumscribed chondrosarcoma in the posterior mediastinum without demonstrable attachment to bone, although rare, is exemplified by the following case.

CASE REPORT

F. E., a 26-year-old white housewife,* was admitted for evaluation of severe interscapular and left thoracic pain of 2 years' duration. The only associated symptoms were insomnia and a 10-pound weight loss due to anorexia, both secondary to the pain. There was no history of previous injury, familial disorder, or significant illness. The onset of pain 2 years previously had been gradual and intermittent, being located in the left interscapular area and more severe in the supine position. For about 1 year prior to admission it had been almost constantly present, tending to assume a radicular pattern on the left side, and had become progressively more intense, making her existence almost intolerable. She had consulted numerous physicians here and in Europe, and, in spite of quite elaborate studies, no organic lesion had been detected. One year prior to admission, x-ray studies of the chest at another hospital showed a definitely abnormal mediastinal shadow, but these roentgenograms

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had been interpreted as normal. Films of the spine and a myelogram done at this time showed no abnormality. Intercostal nerve blocks and an empirical course of x-ray therapy had given no relief, and she had been labeled as psychoneurotic.

Physical examination at this admission showed no abnormality except for slightly diminished breath sounds over the upper left posterior thorax. There were no stigmata of neurofibromatosis, and careful skeletal and neurological examinations were negative. She did not appear acutely or chronically ill.

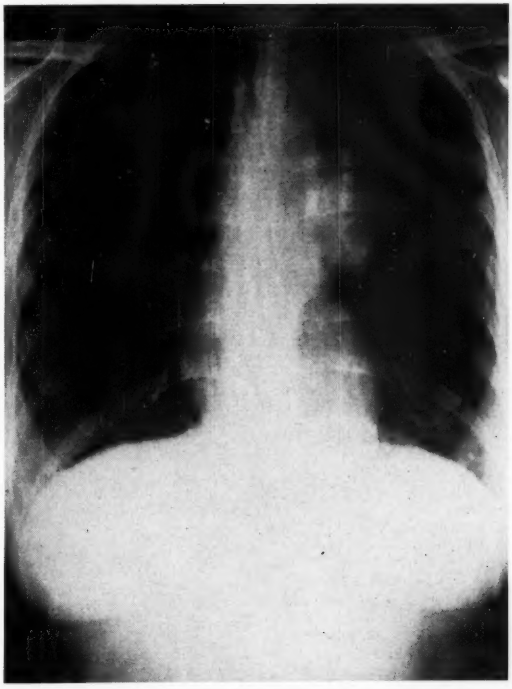

Fig. 1.—Roentgenogram showing large, circumscribed density in mediastinum extending into left hemithorax. Calcifications are visible, especially near the center. A lateral film confirmed its posterior location.

Complete hematologic studies, blood chemical studies, and urinalysis were within normal limits.

Roentgenograms of the chest (Fig. 1) showed a large, circumscribed, irregular density occupying the posterior mediastinum and left paravertebral gutter and extending into the left hemithorax just below the aortic arch. Finely scattered calcific densities were visible within the mass. There was no erosion of the spine. Fluoroscopy demonstrated the mass to be nonpulsating.

Exploratory thoracotomy was performed on June 20, 1957, using a left lateral approach and fifth interspace incision. A solid, circumscribed, bosselated tumor the size of a small grapefruit lay extrapleurally in the left vertebral gutter and posterior mediastinum. It was adjacent to the sixth, seventh, and eighth vertebral bodies and corresponding posterior ribs, and to the descending aorta. The lung, pulmonary hilum, vagus and phrenic nerves were not involved. The mass was rather easily freed from the chest wall, removing periosteum of subjacent ribs with it. The costal cortex was nowhere eroded, and the interspace soft tissue

Fig. 2.—Cut section of excised tumor showing irregular calcific areas about the center surrounded by chondrosarcomatous tissue progressing to the smooth, firm, highly malignant "round cell sarcoma" at the periphery.

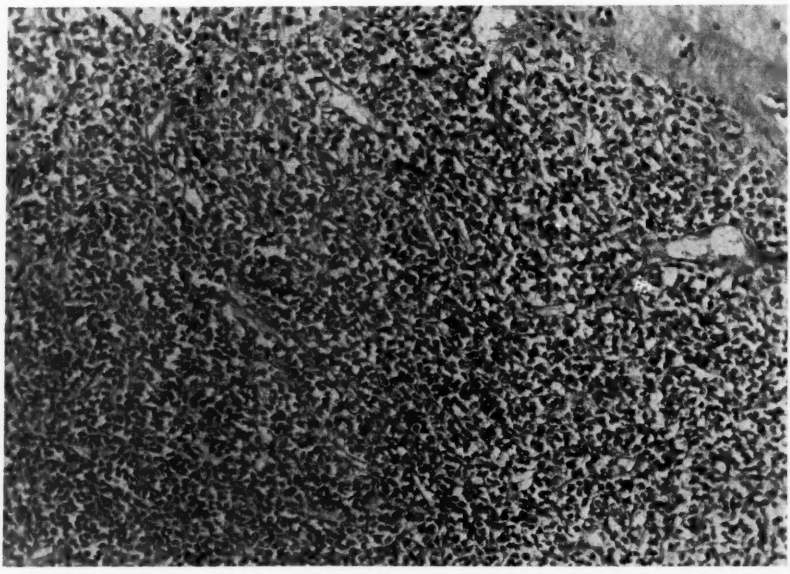

Fig. 3.—Photomicrograph of peripheral portion of tumor. Note highly undifferentiated and cellular tissue. (×200; reduced ¼.)

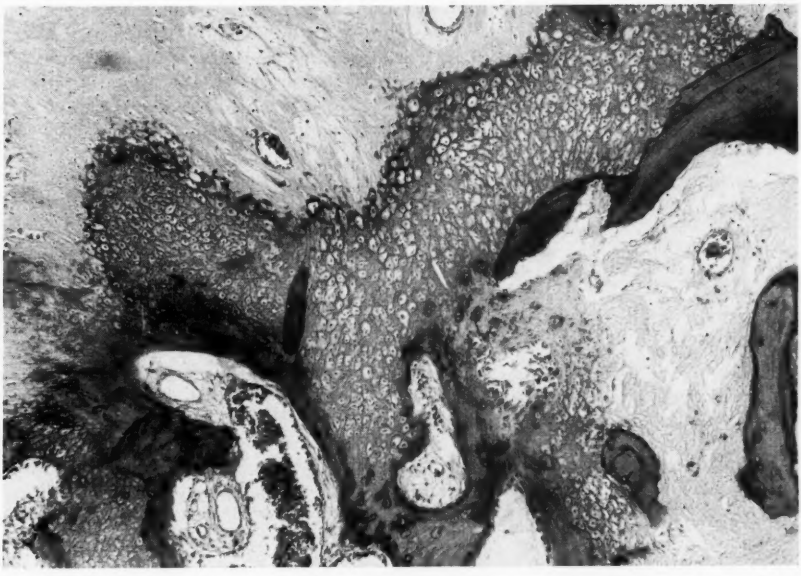

Fig. 4.—Photomicrograph from central portion well-differentiated cartilage and bone. (×100; reduced ¼.)

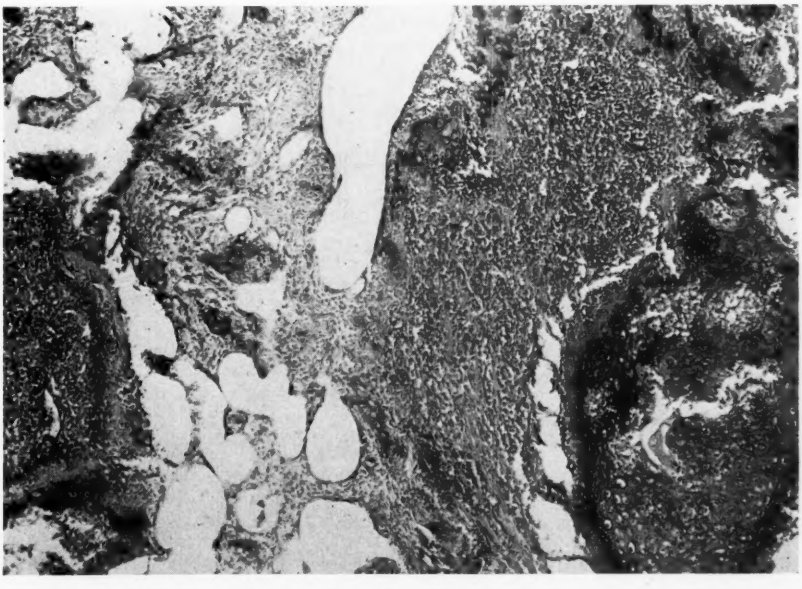

Fig. 5.—Photomicrograph from tumor showing transition zone. Note pleomorphism and irregularity of arrangement of cells. This chondrosarcomatous pattern was found principally between central well-differentiated and peripheral undifferentiated areas. (×100; reduced ¼.)

was not invaded. The mass was carefully separated from the vertebral bodies and their cartilages, to none of which was it adherent, and removed intact. Immediate inspection of the tumor and examination of its bed indicated its apparent complete removal and there was no gross involvement of bone. Convalescence was entirely uneventful, and the patient was discharged on the seventh postoperative day virtually free of pain. At this time, 14 months after operation, she is completely asymptomatic; physical examination is negative; spine and chest films are normal.

Pathologic Findings.—Specimen consisted of a firm, coarsely nodular mass measuring 9 by 6 by 6 cm. The greater portion of the tumor was encapsulated, but a crescentic rim on one surface was smooth and unencapsulated. The cut surface revealed a thick peripheral rim of firm pearly gray to pale yellow tissue and an irregular large central area composed of calcific and dense tissue (Fig. 2). Sections from the periphery of the tumor showed a very cellular and poorly differentiated sarcoma, the histogenesis of which was difficult to identify (Fig. 3). Large sections from the central area revealed an osteochondroma (Fig. 4); this was centrifugally succeeded by a chondrosarcomatous pattern (Fig. 5). This tumor in all probability consisted of an old osteochondroma in which the cartilage had undergone progressive transformation to malignancy as a chondrosarcoma and more recently to a highly undifferentiated sarcoma.

SUMMARY

A chondrosarcoma of the posterior mediastinum without demonstrable origin from bone, consisting of both benign osteochondromatous tissue and highly undifferentiated round cell sarcoma is presented.

Extirpation resulted in complete relief of severe unexplained thoracic pain of 2 years' duration, originally considered to be psychoneurotic in origin.

We are indebted to Dr. Lent Johnson and his staff of the Armed Forces Institute of Pathology, Washington, D. C., for thorough study and confirmation of the pathologic nature of the neoplasm.

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FACTORS INVOLVED IN THE RECOVERY OF A PATIENT AFTER PROLONGED VENTRICULAR FIBRILLATION DURING HYPOTHERMIA

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VENTRICULAR fibrillation is still a formidable complication frequently observed during cardiac surgery performed under hypothermia. In spite of extensive investigation[1] little is known about either the etiology of ventricular fibrillation or the pathophysiologic mechanisms occurring during hypothermia. Because cooling causes almost all tissues to become less irritable, it is paradoxical that the heart becomes more irritable under the same circumstances.

The purpose of this report is to describe a case in which unusually prolonged ventricular fibrillation occurred during the closure of an interatrial septal defect under hypothermia, and in which there was a restoration of normal sinus rhythm only after the body temperature reached 32.5° C.

CASE REPORT

S. K., a 10-year-old white girl, was admitted to National Jewish Hospital on June 3, 1957. The third of four siblings, she was delivered at term after an uncomplicated pregnancy. During routine examination at the age of 18 months, the patient was found to have a cardiac murmur. Development was normal, but the patient had been thin, underweight, easily fatigued, and had frequent respiratory illnesses. She became slightly cyanotic on excitement or overexertion.

Physical examination revealed no clubbing or edema, and there was no cyanosis at rest. Pulse rate was 88 per minute. Neck veins were not distended. The precordium was observed to be slightly overactive. The point of maximum impulse was in the fifth interspace at the midclavicular line. The size of the heart was slightly increased on percussion. A right ventricular thrust was felt along the left sternal border. A Grade 3, harsh, systolic murmur was heard along the left sternal border, loudest at the second left interspace. The murmur was well transmitted to the left shoulder. Though split during both inspiration and expiration, P_2 was of normal intensity. Blood pressure in both arms was 112/36 mm. Hg.

During fluoroscopy (Fig. 1) a marked increase in the pulmonary vascularity was observed. The right ventricle and the right atrium were enlarged. Main, right, and left pulmonary arteries were prominent and overly active. During a barium swallow, the left atrium appeared to be normal in size.

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The electrocardiogram (Fig. 2) revealed right ventricular hypertrophy with prominent R waves in right precordial leads and deep S waves in left precordial leads. The ventricular activation time in Lead V_{3R} measured 0.04 second.

Laboratory data: Urinalysis revealed the specific gravity to be 1.022, reaction 6, negative for sugar albumin and acetone, with 3 to 5 white blood cells per high power field on microscopic examination. Fasting blood sugar was 92 mg. per cent, blood urea nitrogen was 13 mg. per cent. Total protein was 7.3 Gm. per cent with albumin/globulin rate of 5.1 to 2.2. Alkaline phosphatase was 6.2 units, thymol turbidity 2 units, serum glutamic oxaloacetic transaminase 16 units. The cephalin flocculation test was negative at 24 and 48 hours. Bilirubin, 1 minute, was 0.15 mg. per cent. Hematocrit was 49 per cent; white blood count, 10,000 cells per cubic millimeter with a differential of 66 per cent neutrophils and 34 per cent lymphocytes. The red cell sedimentation rate was 10 mm. in 60 minutes.

On June 13, 1957, a right heart catheterization was performed. There was a significant elevation of the oxygen content of blood in the right atrium over blood in the superior vena cava. The elevation persisted throughout the right ventricle and pulmonary arteries. The right pulmonary artery saturation was 74.7 per cent. The oxygen saturation of blood in the brachial artery on exercise decreased to 79.6 per cent. At rest, the pulmonary artery pressure rose to 50/20 mm. Hg, whereas the right ventricular pressure rose to 77/–1 mm. Hg.

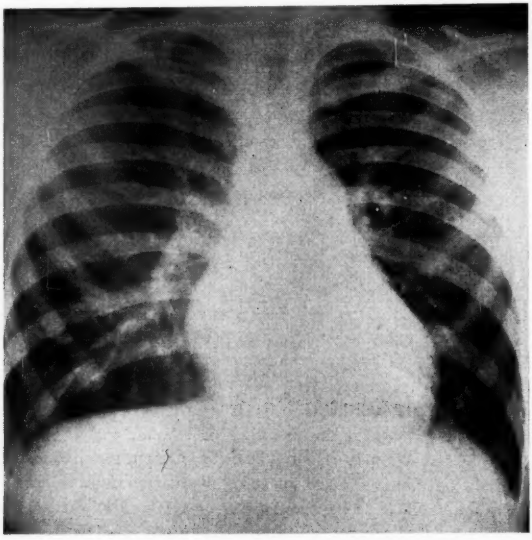

Fig. 1.—Roentgenogram, prior to surgery, showed prominent right atrium, main, right, and left pulmonary arteries, and increased lung vascularity. These are hallmarks of the secundum type atrial septal defect.

The resting systemic flow was 5,400 c.c. per minute, with a cardiac index of 4.7 L. per minute per square meter of body surface. The resting pulmonary flow was 6,600 c.c. per minute, with a cardiac index of 5.7 L. per minute per square meter of body surface. The pulmonary flow rose to 11,000 per minute and the index to 9.5 L. per minute per square meter following exercise. Total pulmonary resistances were 182 dynes per centimeter per second at rest and 189 dynes per centimeter per second on exercise. The superior vena cava oxygen saturation was 64.7 per cent and the saturation of the right atrium at different levels was 73.0 per cent and 74.0 per cent.

The catheterization data were consistent with a left-to-right shunt at the level of the right atrium. The gradient existing between the level of the pulmonary artery and the systolic level of the right ventricle was believed to be secondary to the high pulmonary flow.

On June 28, 1957, under induced hypothermia of 30° C., inflow-outflow occlusion, procaine infiltration of the sino-auricular node, and coronary arterial perfusion of oxygenated heparinized blood, a secundum type, 3 cm., interatrial septal defect was found and closed.

While closing the incision in the right atrium, a tear developed. This began at the lower aspect of the interatrial fissure and extended to the inferior vena cava. There was a moderate blood loss and a delay in re-establishing the temporarily occluded circulation. The time interval from total inflow-outflow occlusion to release of the clamps was 9 minutes, 45 seconds. The rectal temperature during the period of inflow-outflow occlusion was approximately 30° C.

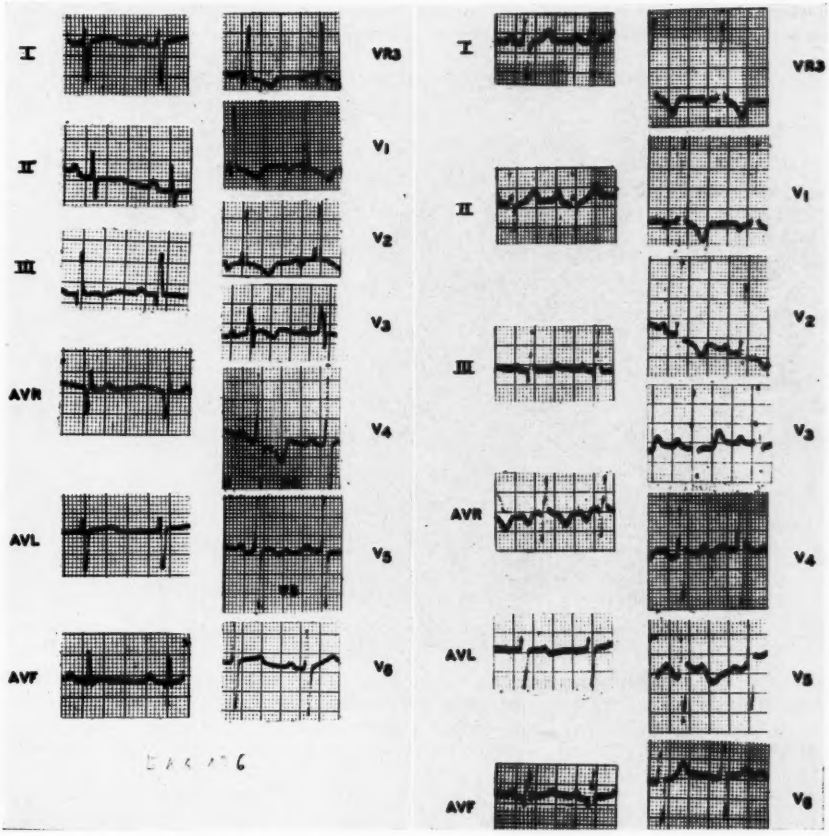

Fig. 2.—Electrocardiograms before and after surgery. The tracings reveal right ventricular outflow tract hypertrophy with no significant change after surgery.

After the atrial bleeding had been controlled, the ventricles started to fibrillate (Fig. 3). This fibrillation resisted eight attempts at electrical defibrillation. On four occasions, 2 c.c. of Adrenalin (1:10,000) were injected into the pulmonary artery without response. During this period the heart was bathed with warm saline solution which was poured into the chest cavity. Circulation was maintained for 60 minutes by intermittent cardiac compression. Care was used to squeeze the heart in the palm of the hand at a rate of approximately 80 per minute. After 60 minutes, when the esophageal temperature rose to 32.5° C.,

a transient normal sinus rhythm was observed. This was soon followed by atrial fibrillation. The aortic arch had been occluded for 1 hour and 18 minutes, during the period of ventricular fibrillation.

The chest was closed in layers, and the sternum was approximated with three interrupted sutures of No. 30 wire. A chest tube was inserted on each side posteriorly at the eighth interspace and a third on the right at the second interspace. At the time of closure of the incision, the patient's rectal temperature was 33.5° C. The patient had been given 1,000 c.c. of blood intravenously and 1,000 c.c. had been injected into the ascending aorta for coronary arterial perfusion.

During the first 36 postoperative hours, systemic hypotension was noted in the patient, who was then treated with Levophed, Neosynephrine, and blood. The urinary output during this time was only 300 c.c. The electrocardiogram 36 hours after surgery revealed an atrial flutter-fibrillation; the ventricular rate was 125 per minute.

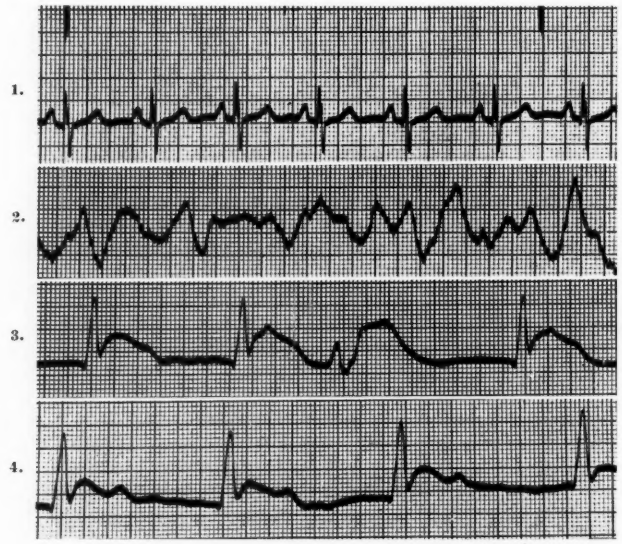

Fig. 3.—Lead II tracings taken during surgery revealed the development of ventricular fibrillation (*2*) and injury complexes following reversion to supraventricular rhythm (*3* and *4*).

On the third postoperative day, the urinary output increased to 1,200 c.c. Blood urea nitrogen was 34 mg. per cent. The rhythm was still atrial flutter-fibrillation. On the fourth postoperative day, the patient's general condition improved further. Urinary output was 1,400 c.c. Because the ventricular rate was rather high, 130 to 140 per minute, the patient was digitalized. On the seventh postoperative day, a regular sinus rhythm was established. At this time, the patient complained of tenderness of back and thigh muscles, and resisted straightening her legs. It was then observed that the deep tendon reflexes were absent in the legs and sluggish in the upper extremities. Abdominal reflexes could not be elicited. Her mental status was normal, and the results of neurological examination were otherwise within normal limits. Spinal fluid pressure was normal; only three lymphocytes were present per cubic centimeter; chloride was 118 mEq./L., protein 30 mg. per cent, glucose 58 mg. per cent, and serum glutamic oxaloacetic transaminase 13 units. It was believed that the patient had an injury to the anterior horn cells of the spinal cord. Complete muscle testing revealed weakness of scattered muscle groups in the back. Extensor and hamstring muscles of the legs were particularly tight. Straight leg raising was limited to 20 degrees.

An active program of muscle re-education was initiated, and by July 29, 1957, she began to walk with the support of a cane. On Aug. 18, 1957, hamstring spasm was minimal and muscle strength had increased. In September, 1957, 3 months after surgery, the patient had made satisfactory progress in gait training and showed good hip stabilization. Moderate muscle weakness persisted in the gastrocnemius group. The posterior tibial muscles were slightly weak, opposed by strong peroneal muscles, resulting in eversion of the feet. Orthopedic shoes were used to correct this condition.

On Aug. 9, 1957, an electroencephalogram revealed widespread cortical irritability. The clinical implications of these findings were not clear. The psychometric studies, which included Bender Gestalt, Draw-A-Person, and Wechsler intelligence scale studies, disclosed that the patient had neither intellectual impairment nor any evidence of organic brain damage. She seemed to be functioning on a superior level in all her activities.

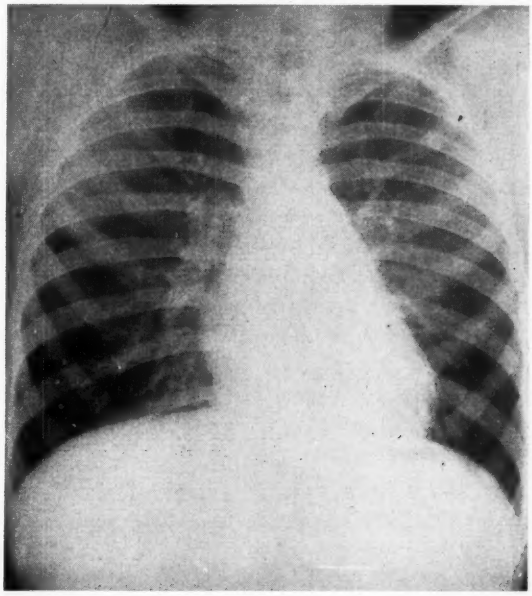

Fig. 4.—Roentgenogram taken 5 months after surgery. The vascularity is less prominent than before surgery. Main, right, and left pulmonary arteries are smaller. Right atrium is smaller. Repeat catheterization revealed the defect to be completely closed.

On Oct. 18, 1957, the patient was discharged, significantly improved in her muscle condition. Discharge examination on Oct. 19, 1957, revealed the pulse to be regular at 80 per minute. The precordium was quiet on inspection and palpation. No thrust or thrills were felt. A Grade 2, blowing, medium-pitched, systolic murmur was audible at the second left intercostal space. P_2 was finely split, mainly during inspiration. The blood pressure was 120/75 mm. Hg. Femoral pulsations were normal. Neck veins were not distended. No edema or cyanosis was noted. Roentgenograms (Fig. 4) showed the over-all size of the heart to be definitely smaller. No changes were noted in the vascularity of the lungs; furthermore, the heart was significantly less active.

Although there was slight residual muscle impairment, the physiatrist expressed the belief that the required program of muscle re-education would be as effective at home as in the hospital.

DISCUSSION

Our observations in this case emphasize that ventricular fibrillation can last for an hour or more with subsequent complete recovery of the patient. Owens[2] described recovery following ventricular fibrillation which lasted 86 minutes during a normothermic operation. Husfeldt and Secher[3] have reported 3 cases of ventricular fibrillation lasting 114, 108, and 50 minutes during hypothermic surgery. (One case of interatrial septal defect and 2 cases of isolated pulmonic stenosis.) Complete recovery was observed in 2 cases and recovery with residual brain damage in the other.

The neurological changes observed in this case were undoubtedly the sequelae of spinal cord damage secondary to cross-clamping of the aorta for 78 minutes. At the present time, there is little information regarding the tolerance of the spinal cord to anoxia. Tolerance is greater in hypothermia, but the available data are equivocal, with estimates ranging from 30 minutes to 2 hours.[4, 5] In our case, the cross-clamping of the aorta was maintained in order to divert blood to the brain during the critical period of ventricular fibrillation and intermittent cardiac compression. The severe oliguria observed during the first 24 hours after surgery may have been a consequence either of the patient's shocklike state or of renal ischemia. It seems likely that the coronary and carotid perfusion with oxygenated blood during the period of total circulatory occlusion, followed by aortic occlusion during the period of ventricular fibrillation, were factors in preventing heart and brain damage.[6]

The extent of myocardial damage in this case was difficult to estimate. Transitory but reversible changes were observed in the electrocardiogram. Damage could have been a consequence of anoxia, or perhaps secondary to cardiac compression. It is known that there is a wide range of pathologic alterations in the heart following cardiac compression and that these changes may involve any or all of the cardiac tissues. The vascular structures within the myocardium and pericardium are most vulnerable, and usually there is a hemorrhagic extravasation of blood. Gross laceration and even perforation of the myocardium are not infrequent. It should be re-emphasized that the most important factor in avoiding such complications is the skill with which intermittent cardiac compression is performed.[1] Care must be exercised that one applies pressure with the palm of the hand instead of the fingers.

At present, the incidence of ventricular fibrillation is reasonably low during operations involving the atrial wall or pulmonary artery.[7] Cutting of the ventricle increases the incidence of ventricular fibrillation. However, as this case emphasizes, ventricular fibrillation can occur in cases in which the ventricles are not incised. When fibrillation does occur during hypothermia, its control is more difficult than during normothermia. There is general agreement that the temperature level should not drop below 28° to 30° C. Temperatures below this are attended by prohibitive incidence of ventricular fibrillation. Certain rather obvious, although at times forgotten, technical errors, such as an

inaccurate thermometer, should be studiously avoided. When fibrillation does occur, rewarming of the heart is usually necessary before electric shock will permanently revert the rhythm to one of supraventricular origin.

There is no uniform opinion concerning the rapidity of cooling. The rapidity varies according to the method used (surface cooling or extracorporeal technique) but, in a medium-sized adult, a fall in temperature of 1° C. each 6 to 8 minutes seems to be advisable and this approach was applied in this case.

It has been shown[8] that sino-auricular blockage with Procaine is effective in preventing ventricular fibrillation in cooled dogs. This procedure was employed in our case. There are not enough data available to establish the validity of this procedure. Also, recommended, although not used in this case, is the administration of Prostigmin with coronary perfusion.[9] Because Prostigmin is a cholinesterase inhibitor, it is assumed that its antifibrillatory action is secondary to an accumulation of acetylcholine. A heart arrested by acetylcholine has been supposed to be less susceptible to subsequent hypothermic ventricular fibrillation.

Some authorities believe that infusions of dextrose, amino acids, or fat intravenously, before and during hypothermia, reduce the incidence of fibrillation. It is not known whether these solutions act by alterations in the ion partition or by providing more nutrient to the heart. Dextrose was administered intravenously prior to surgery in this patient.

Careful electrocardiogram monitoring may be of value in detecting impending disaster. Loss of P waves, wandering pacemaker, slow rate, prolonged Q-T interval, and low voltage T waves are common and to be expected. However, marked prolongation of the QRS complexes with severe S-T segment depression herald ventricular fibrillation and demand immediate cessation of the hypothermia and warming of the patient. It is interesting that in this patient the ventricular fibrillation occurred suddenly without any electrocardiographic signs of warning.

SUMMARY

A case in which prolonged ventricular fibrillation occurred following the closure of a secundum type interatrial septal defect under hypothermia and coronary arterial perfusion is reported. The ventricular arrhythmia lasted 60 minutes, followed by recovery of the patient. Aortic occlusion above the left subclavian artery was maintained for 78 minutes in order to divert the blood flow to the brain and the heart. The only observed neurological sequelae in this case were attributed to spinal cord ischemia resulting from prolonged occlusion of the aorta. Coronary and carotid arterial perfusion combined with cross-clamping of the aorta no doubt lessened the extent of brain and myocardial damage in this case. The ventricular fibrillation reverted to normal sinus rhythm after repeated electric shocks when the heart was warmed to 32.5° C. Such recovery after prolonged ventricular fibrillation should encourage surgeons to persist zealously in attempts to defibrillate.

The authors are indebted to George C. Heitzman, M.D., presently of Syracuse, N. Y., and Charles V. Demong, M.D., of Denver, Colo., who performed the surgery in the case. Goffredo Gensini, M.D., Chief of Cardiopulmonary Physiology at National Jewish Hospital, performed the catheterization of this patient. Baruch Bromberger-Barnea, Ph.D., Department of Research and Laboratories, kindly reviewed the manuscript of this report and offered many valuable suggestions.

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Erratum

In the September issue of the JOURNAL, on page 325, Dr. Irving A Sarot's discussion of the three preceding papers was wrongly attributed to Dr. Irving A. Short. We regret exceedingly the misspelling of Dr. Sarot's name.

Announcements

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The Thirty-ninth Annual Meeting of the American Association for Thoracic Surgery will be held April 21, 22, and 23, 1959, in Los Angeles, California. Headquarters will be the Hotel Statler.

Requests for Hotel Reservations

These should be mailed directly to Mr. William Truman, Reservation Manager, Hotel Statler, Los Angeles, California. Please mention this Association, the type of accommodation desired, the date, and approximate hour of arrival and departure. If accommodations are desired elsewhere in Los Angeles, please communicate directly with the hotel of your choice.

Material for the Program

SCIENTIFIC PAPERS.—Abstracts of all papers proposed for presentation at the 1959 meeting *must be received on or before November 15, 1958,* otherwise they will not be considered by the Program Committee. These should be labeled "For Thoracic Surgery Forum" or "For Regular Program" depending upon the nature of the subject matter, in the same manner as in previous years. They should contain from 200 to 250 words that accurately reflect the content of the completed paper.

Six, repeat, six clearly legible copies of *each* abstract must be sent to Miss Ada Hanvey, Administrative Assistant, The American Association for Thoracic Surgery, Suite 308, Carondelet Building, 7730 Carondelet Avenue, St. Louis 5, Missouri.

Essayists selected for the program are reminded that papers presented at the meeting *must* be handed to the secretary *immediately* after their presentation. If they are not ready at that time there is a chance that they will not be published in THE JOURNAL OF THORACIC SURGERY.

MOTION PICTURE SESSION.—Plans are again being made to show suitable motion pictures selected on a competitive basis. *The films themselves,* together with descriptive material concerning length of presentation, etc., must be received by Dr. John C. Jones, Suite 509, 1136 West Sixth Street, Los Angeles 17, California, *before November 15, 1958.*

Applications for Membership

Applications for Associate Membership in the Association must be received by the Membership Committee ***not later than November 15, 1958,*** otherwise the application will be deferred for consideration until the 1960 meeting.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the signature on the application form, the sponsors will forward a *separate* letter concerning the applicant directly to:

Dr. Duane Carr
Chairman of the Membership Committee
899 Madison Avenue
Memphis 3, Tennessee

Sponsors are reminded that *new* letters of recommendation must be forwarded to the Chairman of the Membership Committee should their applicants be held over for reconsideration a *second year.*